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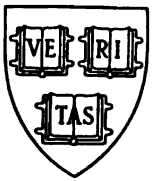

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MEDICO-CHIRURGICAL TRANSACTIONS.

PUBLISHED BY

THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON.

VOLUME THE FORTY-SECOND.

LONDON:
LONGMAN, GREEN, LONGMAN, AND ROBERTS,
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1859.

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1805. WILLIAM SAUNDERS, M.D.
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1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
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1815. HENRY CLINE.
1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, Bart., K.C.H., D.C.L.
1821. JOHN COOKE, M.D.
1823. JOHN ABERNETHY.
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The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed.

OCTOBER 1859.

Amongst the non-residents, those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1841 *James Abercrombie, M.D., Cape of Good Hope.

1846 *John Abercrombie, M.D., Physician to the Cheltenham General Hospital, 13, Suffolk square, Cheltenham.

1851 *Henry Wentworth Acland, M.D., F.R.S., Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 Elias Acosta, M.D., New York, U.S.

1842 William Acton, 46, Queen Anne street, Cavendish square. *Trans.* 1

1851 John Adams, Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, the London Hospital; 4, St. Helen's place, Bishopsgate street. *Trans.* 1.

Elected

1852 WILLIAM ADAMS, Surgeon to the Royal Orthopædic Hospital; Lecturer on Anatomy and Surgery at the Grosvenor place School of Anatomy and Medicine; 5, Henrietta street, Cavendish square. *Trans.* 2.

1818 THOMAS ADDISON, M.D., Senior Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital, and Consulting Physician to the Royal South London Dispensary; 51, Berkeley square. C. 1826, 1853. V.P. 1837. P. 1849-50. *Trans.* 2.

1837 *RALPH FAWSETT AINSWORTH, M.D., Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1819 GEORGE FREDERICK ALBERT.

1839 RUTHERFORD ALCOCK, K.C.T., K.T.S., H.M.'s Consul-General in Japan. *Trans.* 1.

1826 JAMES ALDERSON, M.D., F.R.S., Senior Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 17, Berkeley square. S. 1829. C. 1848. T. 1849. V.P. 1852-3. *Trans.* 3.

1843 CHARLES JAMES BERRIDGE ALDIS, M.D., Medical Officer of Health for St. George's, Hanover square; Senior Physician to the Surrey Dispensary; and Physician to the St. Paul and St. Barnabas Dispensary; 1, Chester terrace, Chester square.

1850 CHARLES REVANS ALEXANDER, Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork street, Bond street.

1836 HENRY ANCELL, 3, Norfolk crescent, Oxford square. C. 1847-8. *Trans.* 2.

1820 THOMAS ANDREWS, M.D., Norfolk, Virginia.

1819 PROFESSOR ANTOMMARCHI, Florence.

1819 JAMES MONCRIEFF ARNOTT, F.R.S., Surgeon in Ordinary to H.R.H. the Prince Consort, President of the Royal College of Surgeons; 2, New Burlington street. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.

Elected

1817 JOHN ASHBURNER, M.D., F.L.S., Hyde Park place, Cumberland gate. C. 1821, 1830-1.

1851 THOMAS JOHN ASHTON, Surgeon to the Blenheim street Dispensary; 31, Cavendish square.

1825 BENJAMIN GUY BABINGTON, M.D., F.R.S., Physician to the Asylum for Deaf and Dumb, and Consulting Physician to the German Hospital, and to the City of London Hospital for Diseases of the Chest; 31, George street, Hanover square. C. 1829. V.P. 1845-6. T. 1848. *Trans.* 2.

1846 CORNELIUS METCALFE STUART BABINGTON, F.R.C.P., Physician to Queen Charlotte's Lying-in Hospital, and Assistant-Physician to the Hospital for Sick Children; 29, Hertford street, May fair. C. 1859.

1820 *JOHN H. BADLEY, Dudley, Worcestershire.

1838 FRANCIS BADGLEY, M.D., 43, Albion street, Hyde park.

1840 WILLIAM BAINBRIDGE, late of Kingston, Surrey.

1836 ANDREW WOOD BAIRD, M.D., Physician to the Dover Hospital; Dover, Kent.

1851 *ALFRED BAKER, Surgeon to the Birmingham General Hospital, and Lecturer on Surgery at Sydenham College; Congreve street, Birmingham.

1839 THOMAS GRAHAM BALFOUR, M.D., F.R.S., Deputy Inspector-General of Hospitals; 10, Sumner place, Onslow square, Brompton. C. 1852-3. *Trans.* 1.

1848 EDWARD BALLARD, M.D., Medical Officer of Health for Islington; 42, Myddelton square. *Trans.* 1.

1849 THOMAS BALLARD, 10, Southwick place, Hyde park.

1837 WILLIAM BALY, M.D., F.R.S., *Treasurer*, Physician Extraordinary to H.M. the Queen; Assistant-Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 45, Queen Anne street, Cavendish square. C. 1845-6. L. 1847. S. 1848-9. V.P. 1855-6. *Trans.* 1.

Elected

1847 ANDREW WHYTE BARCLAY, M.D., *Secretary*, Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-8. *Trans.* 2.

1848 EDGAR BARKER, 9, Oxford square, Hyde park.

1833 THOMAS ALFRED BARKER, M.D., Senior Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 71, Grosvenor street. C. 1844-5. V.P. 1853-4. *Trans.* 5.

1843 THOMAS HERBERT BARKER, M.D., Harpur place, Bedford.

1847 GEORGE HILARO BARLOW, M.D., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; Physician to the Magdalen Hospital; 5, Union street, Southwark. C. 1859.

1840 BENJAMIN BARROW, Surgeon to the Royal Isle of Wight Infirmary; Clifton House, Ryde, Isle of Wight.

1844 WILLIAM RICHARD BASHAM, M.D., Senior Physician to, and Lecturer on Medicine at, the Westminster Hospital; 17, Chester street, Grosvenor place. S. 1852-4. *Trans.* 2.

1841 GEORGE BEAMAN, M.D., 3, Henrietta street, Covent garden.

1856 AMOS BEARDSLEY, Ulverstone, Lancashire.

1836 WILLIAM BEAUMONT, Professor of Surgery in the University of King's College, Toronto, Upper Canada. *Trans.* 2.

1840 CHARLES BEEVOR, 41, Upper Harley street.

1858 WILLIAM CHAPMAN BEGLEY, M.D., Middlesex County Lunatic Asylum; Hanwell.

1819 THOMAS BELL, F.R.S., F.L.S., Professor of Zoology in King's College, London; Surgeon-Dentist to, and Lecturer on the Anatomy and Diseases of the Teeth at, Guy's Hospital; and President of the Linnean Society; 17, New Broad street, City. C. 1832-3. V.P. 1854. *Trans.* 1.

1847 JAMES HENRY BENNET, M.D., Physician-Accoucheur to the Royal Free Hospital.

1845 EDWIN UNWIN BERRY, 7, James street, Covent garden.

1820 STEPHEN BERTIN, Paris.

Elected

1815 ARCHIBALD BILLING, M.D., F.R.S., late Senior Physician to the London Hospital; Member of the Senate, and Examiner in Medicine at the University of London; 6, Grosvenor gate. C. 1825. V.P. 1828-9.

1827 WILLIAM BIRCH, Barton-under-Needwood, Staffordshire. *Trans.* 2.

1850 JAMES BIRD, M.D., Lecturer on Military Surgery at St. Mary's Hospital Medical School; 27, Hyde park square.

1855 PETER HINCKES BIRD, 1, Norfolk square, Hyde park.

1856 WILLIAM BIRD, Surgeon to the West of London Hospital, and the St. George's and St. James's Dispensary; 11, George street, Hanover square.

1849 EDMUND LLOYD BIRKETT, M.D., Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square.

1851 GEORGE BIRKETT, M.D., Lecturer on Medical Jurisprudence at the Charing Cross Hospital; Northumberland House, Green lanes, Stoke Newington.

1851 JOHN BIRKETT, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 59, Green street, Grosvenor square. L. 1856-7. *Trans.* 4.

1846 HUGH BIRT, 12, High street, Portsmouth.

1843 PATRICK BLACK, M.D., Assistant-Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 49, Queen Anne street, Cavendish square. C. 1856.

1847 GEORGE C. BLACKMAN, M.D., Professor of Surgery in the Medical College of Ohio; New York, U.S.

1839 RICHARD BLAGDEN, Surgeon-Accoucheur, and Surgeon Extraordinary to H.M. the Queen, and Surgeon in Ordinary to H.R.H. the Duchess of Kent; 7, Percy place, Walcot, Bath. C. 1847-8. *Trans.* 1.

1840 PEYTON BLAKISTON, M.D. F.R.S., St. Leonard's-on-Sea.

1845 HENRY BLENKINSOP, Senior Surgeon to the Warwick Dispensary; Jury street, Warwick.

1823 LOUIS HENRY BOJANUS, M.D., Wilna.

Elected

1846 PETER BOSSEY, 1, Queen's terrace, The Common, Woolwich, Kent, and Worthing, Sussex.

1846 JOHN ASHTON BOSTOCK, Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 54, Chester square, Belgravia.

1841 WILLIAM BOWMAN, F.R.S., Surgeon to King's College Hospital, and to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. *Trans.* 3.

1857 WILLIAM BRINTON, M.D., Physician to the Royal Free Hospital, and Lecturer on Physiology and Forensic Medicine at St. Thomas's Hospital; 20, Brook street, Grosvenor square.

1851 BERNARD EDWARD BRODHURST, Assistant-Surgeon to the Royal Orthopædic Hospital, and Surgeon to the Hon. Artillery Company; 20, Grosvenor street. *Trans.* 1; *Pro.* 1.

1813 SIR BENJAMIN COLLINS BRODIE, Bart., D.C.L., F.R.S., President of the Royal Society, President of the Medical Council; Serjeant-Surgeon to H.M. the Queen, Surgeon in Ordinary to H.R.H. the Prince Consort; Foreign Correspondent of the Institute of France, and Foreign Associate of the Imperial Academy of Medicine of Paris; 14, Savile row, Burlington gardens. C. 1814-15, 1818-19, 1821-2, 1835-6, 1841-2, 1849. V.P. 1816-17. P. 1839-40. *Trans.* 11.

1844 CHARLES BROOKE, M.A., F.R.S., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 29, Keppel street, Russell square. C. 1855.

1848 WILLIAM PHILPOT BROOKES, M.D., Surgeon to the Cheltenham General Dispensary, and Medical Inspector of Lunatic Asylums for the Upper Division of Gloucestershire; Sherborne Lodge, Cheltenham.

1854 *HENRY BROWN, Surgeon to H.M. the Queen, H.R.H. the Prince Consort, and the Royal Household; Windsor.

1857 *ROBERT BROWN, Surgeon to the Carlisle Dispensary; 4, Devonshire street, Carlisle.

Elected

1851 ALEXANDER BROWNE, M.D., Army and Navy Club, St. James's square; and Twynholm, Kirkcudbright.

1855 WALTER JOHN BRYANT, 7, Bathurst street, Hyde park gardens.

1823 B. BARTLET BUCHANAN, M.D.

1843 JOHN CHARLES BUCKNILL, M.D., Medical Superintendent of the Devon County Lunatic Asylum, Exminster, Devonshire.

1839 GEORGE BUDD, M.D., F.R.S., Professor of Medicine in King's College, London; Physician to King's College Hospital; Consulting Physician to the Seamen's Hospital Ship 'Dreadnought,' and to the Blenheim Free Dispensary; 20, Dover street, Piccadilly. C. 1846-7. V.P. 1857. *Trans.* 5.

1839 THOMAS HENRY BURGESS, M.D.

1853 PATRICK BURKE, 13, Upper Montagu street, Montagu square.

1854 PHILIP BURROWES, Surgeon to the London City Mission; 23, Gloucester crescent north, Hyde park.

1833 GEORGE BURROWS, M.D., F.R.S., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V.P. 1849-50. *Trans.* 2.

1820 SAMUEL BURROWS.

1837 GEORGE BUSK, F.R.S., F.L.S., Surgeon to the Seamen's Hospital Ship 'Dreadnought'; 15, Harley street, Cavendish square. C. 1847-8. V.P. 1855. *Trans.* 4.

1818 JOHN BUTTER, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Plymouth.

1851 *WILLIAM CADGE, Surgeon to the Norfolk and Norwich Hospital; All Saints, Norwich. *Trans.* 1.

1851 THOMAS CALLAWAY, India.

1852 *GEORGE CANNEY, Bishop-Auckland, Durham.

1847 JOHN BURFORD CARLILL, Surgeon-Accoucheur to the Newman street Lying-in Institution; 57, Berners street.

1825 HARRY W. CARTER, M.D., F.R.S., Edinb., Consulting Physician to the Kent and Canterbury Hospital; Ashford, Kent.

Elected

1853 ROBERT BRUDENELL CARTER, Newthorpe, Nottinghamshire.

1820 SAMUEL CARTWRIGHT, F.R.S., F.L.S., Nizell's House, near Tunbridge, Kent.

1845 SAMUEL CARTWRIGHT, Jun., Surgeon-Dentist to King's College Hospital; 32, Old Burlington street.

1839 WILLIAM CĀTHROW, 42, Weymouth street, Portland place.

1845 WILLIAM OLIVER CHALK, Surgeon to the St. Marylebone Eye and Ear Institution; 3, Nottingham terrace, York gate, Regent's park [40, Marylebone road].

1844 THOMAS KING CHAMBERS, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Physician to the Lock Hospital; 1, Hill street, Berkeley square. *Trans.* 1.

1849 FREDERICK CHAPMAN, Richmond green, Surrey.

1837 HENRY THOMAS CHAPMAN, 16, Lower Seymour street, Portman square. C. 1858.

1852 GEORGE BOBLASE CHILDS, Surgeon-in-Chief to the City Police Force, and Surgeon to the Metropolitan Free Hospital; 11, Finsbury place South.

1842 WILLIAM DINGLE CHOWNE, M.D., Physician to, and Lecturer on Medicine and Midwifery at, the Charing Cross Hospital; Corresponding Fellow of the Royal Academy of Surgery of Madrid; 8, Connaught place West, Hyde park. C. 1853-4.

1839 FREDERICK LE GROS CLARK, Surgeon to, and Lecturer on Surgical Anatomy at, St. Thomas's Hospital; Surgeon to the Magdalen Hospital; Consulting Surgeon to the Western General Dispensary, and to the London Female Penitentiary, Pentonville; 14, St. Thomas's street, Southwark, and Lee, Kent. S. 1847-9. V.P. 1855-6. *Trans.* 3.

1845 JOHN CLARK, M.D., Staff Surgeon, 1st Class. Canada.

1848 JOHN CLARKE, M.R.C.P., Physician to the British Lying-in Hospital; 42, Hertford street, Mayfair.

1850 JOSIAH CLARKSON, New Hall street, Birmingham. *Trans.* 1.

1842 OSCAR MOORE PASSEY CLAYTON, 87, Harley street.

Elected

1853 JOSEPH THOMAS CLOVER, 3, Cavendish place, Cavendish square.

1857 CHARLES COATES, F.R.C.P., Edinb., Physician to the Bath General Hospital; 10, Circus, Bath.

1851 EDWARD COCK, Senior Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; 13, St. Thomas's street, Southwark. C. 1857. *Trans.* 3.

1850 DANIEL WHITAKER COHEN, M.D., Headley grove, near Dorking.

1835 *WILLIAM COLBORNE, Chippenham, Wiltshire.

1818 ROBERT COLE, F.L.S., Holybourne, Hampshire.

1855 FREDERICK COLLINS, M.D., Wanstead, Essex.

1828 JOHN CONOLLY, M.D., D.C.L., Consulting Physician to the Middlesex County Lunatic Asylum, Hanwell.

1840 *WILLIAM ROBERT COOKE, Burford, Oxfordshire.

1819 GEORGE COOPER, Brentford, Middlesex.

1841 GEORGE LEWIS COOPER, Surgeon to the Bloomsbury Dispensary; 7, Woburn place, Russell square.

1843 WILLIAM WHITE COOPER, Surgeon-Oculist in Ordinary to H.M. the Queen; Senior Surgeon to the North London Eye Infirmary; and Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Mary's Hospital; 19, Berkeley square. C. 1858-9.

1854 CHARLES THOMAS COOTE, M.D., Assistant-Physician to, and Lecturer on Medical Jurisprudence at, the Middlesex Hospital; 1, Gloucester place, Hyde park.

1841 HOLMES COOTE, Assistant-Surgeon to St. Bartholomew's Hospital, and to the Royal Orthopædic Hospital; 26, New Bridge street, Blackfriars. S. 1853-4. *Trans.* 1.

1835 GEORGE FORD COPELAND, Cheltenham.

1822 JAMES COPLAND, M.D., F.R.S., Consulting Physician to the Royal Infirmary for Children, and to the Great Northern Hospital, King's Cross; Hon. Fellow of the Royal Academy of Sciences of Sweden, &c.; 5, Old Burlington street. C. 1831. V.P. 1838-9. P. 1853-4.

Elected

1847 JOHN ROSE CORMACK, M.D., F.R.S. Edinb., 27, Ampthill square, Hampstead road.

1839 *CHARLES CÆSAR CORSELLIS, M.D., F.L.S., Benson, Oxon.

1853 WILLIAM GILLETT CORY, M.D., Burgh Heath, Sutton, Surrey.

1847 RICHARD PAYNE COTTON, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 46, Clarges street, Piccadilly.

1828 WILLIAM COULSON, Senior Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.

1841 MERVYN ARCHDALL NOTT CRAWFORD, M.D., Wiesbaden. C. 1853-4.

1847 GEORGE CRITCHETT, Senior Assistant-Surgeon to, and Lecturer on Surgery at, the London Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 46, Finsbury square. *Trans.* 1.

1837 JOHN FARRAR CROOKES, Harewell, near Faversham, Kent.

1849 *WILLIAM EDWARD CROWFOOT, Beccles, Suffolk.

1851 JAMES CAMERON CUMMING, M.D., 1, Cadogan place, Sloane street.

1846 HENRY CURLING, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

1837 THOMAS BLIZARD CURLING, F.R.S., *Vice President;* Surgeon to, and Lecturer on Surgery at, the London Hospital; Examiner in Surgery at the University of London; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. *Trans.* 12; *Pro.* 1.

1847 JOHN EDMUND CURREY, M.D., Lismore, County Waterford.

1836 GEORGE CURSHAM, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, and to the Female Orphan Asylum; and Inspector of Anatomy for the Provinces; 55, Victoria street, Westminster. S. 1842-7. C. 1850-1. V.P. 1855. T. 1856-8.

1822 CHRISTOPHER JOHN CUSACK, Chateau d'Eu, France.

Elected

1852 THOMAS CUTLER, M.D., Acting Physician at the Spa Waters; Spa, Belgium.

1828 ADOLPHE DALMAS, M.D., Paris.

1836 *JAMES STOCK DANIEL, Ramsgate, Kent.

1820 GEORGE DARLING, M.D., 6, Russell square. C. 1841-2.

1818 *SIR FRANCIS SACHEVEREL DARWIN, Knt., M.D., Deputy-Lieutenant of Derbyshire; Breadsall Priory, near Derby.

1848 HENRY DAUBENY, 40, York place, Portman square.

1846 FREDERICK DAVIES, 19, Upper Gower street, Bedford square.

1818 HENRY DAVIES, M.D., Consulting-Physician to the British Lying-in Hospital; 6, Duchess street, Portland place. C. 1827-8. V.P. 1848-9.

1847 JOHN DAVIES, M.D., Physician Extraordinary to the Hertford General Infirmary, and Visiting Physician to the Hadham Palace Lunatic Asylum, Hertford.

1853 ROBERT COKER NASH DAVIES, Rye, Sussex.

1852 WILLIAM DAVIES, M.D., Senior Physician to the Bath United Hospital; 10, Gay street, Bath.

1852 JOHN HALL DAVIS, M.D., Physician to the Royal Maternity Charity; Physician-Accoucheur to the St. George's and St. James's Dispensary; and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 11, Harley street, Cavendish square.

1820 THOMAS DAVIS, 28, Spring gardens. C. 1837, 1843.

1818 JAMES DAWSON, Liverpool.

1847 GEORGE EDWARD DAY, M.D. F.R.S., Chandos Professor of Anatomy, and Examiner in Medicine in the University of St. Andrew's.

1858 TEOFILO DELIMA, M.D., Caracas, Venezuela, South America.

1846 *SAMUEL BEST DENTON, Ivy Lodge, Hornsea, East Riding, Yorkshire.

1844 ROBERT DICKSON, M.D. F.L.S., Physician to the Scottish Hospital, and to the British Orphan Asylum, Clapham; 16, Hertford street, May fair.

Elected

1839 JAMES DIXON, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Consulting Ophthalmic Surgeon to the Asylum for Idiots; 2, Portman square. L. 1849-55. V.P. 1857-8. *Trans.* 4.

1845 JOHN DODD.

1857 ARCHIBALD DOUGLAS, M.D., 8, Clifton place, Sussex square, Hyde park.

1853 ROBERT DRUITT, M.R.C.P., Medical Officer of Health for St. George's, Hanover square; 37, Hertford street, May fair. *Trans.* 2.

1846 JOHN DRUMMOND, Deputy-Inspector of Fleets and Hospitals; Melville Naval Hospital, Chatham, Kent. *Trans.* 1.

1845 GEORGE DUFF, M.D., High street, Elgin.

1845 EDWARD WILLSON DUFFIN, 14, Langham place. *Trans.* 1.

1833 ROBERT DUNN, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

1843 CHRISTOPHER MERCER DURRANT, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 HENRY SUMNER DYER, M.D., 37, Bryanston sq. C. 1854-5.

1836 JAMES WILLIAM EARLE, late of Norwich.

1824 GEORGE EDWARDS.

1823 CHARLES CHANDLER EGERTON, Kendall Lodge, Epping.

1848 GEORGE VINER ELLIS, Professor of Anatomy in University College, London; University College, London. *Trans.* 2.

1854 *JAMES ELLISON, M.D., Surgeon in ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1835 WILLIAM ENGLAND, M.D., Ipswich, Suffolk.

1842 JOHN ERICHSEN, Professor of Surgery in University College, London, and Surgeon to University College Hospital; 48, Welbeck street, Cavendish square. C. 1855-6. *Trans.* 2.

1836 GEORGE FABIAN EVANS, M.D., Physician to the General Hospital, Birmingham.

1815 *GRIFFITH FRANCIS DORSETT EVANS, M.D., St. Mary's, Bedford, C. 1838.

1845 WILLIAM JULIAN EVANS, M.D., Pinner, Middlesex.

1858 RANDLE WILBRAHAM FALCONER, M.D., Physician to the Bath United Hospital; 22, Bennett street, Bath.

Elected

1844 ARTHUR FARRE, M.D. F.R.S, Professor of Midwifery in King's College, London, and Physician for the Diseases of Women and Children, to King's College Hospital; 12, Hertford street, May fair. C. 1857.

1831 ROBERT FERGUSON, M.D., Physician Extraordinary to H.M. the Queen, and Consulting Physician to King's College Hospital; 125, Park street, Grosvenor square. C. 1839. V.P. 1847.

1841 WILLIAM FERGUSSON, F.R.S., Surgeon Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince Consort; Professor of Surgery in King's College, London, and Surgeon to King's College Hospital; Examiner in Surgery at the University of London; 16, George street, Hanover square. C. 1849-50. *Trans.* 4.

1852 *ALFRED GEORGE FIELD, 28, Old Steine, Brighton.

1849 GEORGE TUPMAN FINCHAM, M.D., Physician to, and Lecturer on Medical Jurisprudence at, the Westminster Hospital; 2, Eccleston terrace south, Eccleston square.

1836 SIR JOHN WILLIAM FISHER, Surgeon-in-Chief to the Metropolitan Police Force; 5, Grosvenor gate. C. 1843-4.

1838 GEORGE LIONEL FITZMAURICE, 97, Gloucester place, Portman square.

1842 THOMAS BELL ELCOCK FLETCHER, M.D., Physician to the Birmingham General Hospital; Waterloo street, Birmingham. *Trans.* 1.

1859 WILLIAM HENRY FLOWER, Assistant-Surgeon to the Middlesex Hospital; 15, Queen Anne street. *Trans.* 1.

1848 JOHN GREGORY FORBES, Surgeon to the Metropolitan Convalescent Institution; 9, Devonport street, Hyde park. *Trans.* 2.

1852 JOHN COOPER FORSTER, Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Royal Infirmary for Women and Children; 11, Wellington street, Southwark. *Pro.* 1.

1820 THOMAS FORSTER, M.D.

1858 WILSON FOX, M.D., Physician to the North Staffordshire Infirmary; Newcastle-under-Lyme, Staffordshire.

Elected

1856 JOHN F. FRANCE, Lecturer on Ophthalmic Surgery at Guy's Hospital, and Surgeon to the Eye Infirmary attached to the Hospital; 24, Bloomsbury square.

1816 JOHN W. FRANCIS, M.D. LL.D., Professor of Materia Medica in the University of New York, U.S.

1841 JOHN CHRISTOPHER AUGUSTUS FRANZ, M.D., 11, Old Steine, Brighton.

1843 PATRICK FRASER, M.D., Physician to the London Hospital, and to the London Dispensary; 63, Grosvenor street.

1836 JOHN GEORGE FRENCH, Surgeon to the St. James's Infirmary; 41, Great Marlborough street. C. 1852-3.

1849 ROBERT TEMPLE FRERE, M.A., F.R.C.P., Physician-Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; 9, Queen street, May fair.

1846 HENRY WILLIAM FULLER, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 13, Manchester square. *Trans.* 2.

1815 *GEORGE FREDERICK FURNIVALL, Medical Attendant of Great Foster House Asylum for Lunatics; Egham, Surrey.

1854 ALFRED BARING GARROD, M.D. F.R.S., Professor of Materia Medica, Therapeutics, and Clinical Medicine in University College, London, and Physician to the University College Hospital; 84, Harley street, Cavendish square. *Trans.* 8.

1857 GEORGE GREEN GASCOYEN, Assistant-Surgeon to the Lock Hospital, and Lecturer on Descriptive and Surgical Anatomy in the St. Mary's Hospital Medical School; 25, Oxford terrace, Hyde park. *Trans.* 1.

1851 GEORGE GASKOIN, 3, Westbourne park.

1819 HENRY GAULTER.

1848 JOHN GAY, Senior Surgeon to the Great Northern Hospital; 10, Finsbury place South.

1821 *RICHARD FRANCIS GEORGE, Senior Surgeon to the Bath General Hospital; 10, Royal Crescent, Bath.

1854 BERNARD GILPIN, Belle Vue House, Ulverstone, Lancashire.

Elected

1858 BENJAMIN GODFREY, M.D., Carlton House, Enfield, Middlesex.

1851 STEPHEN JENNINGS GOODFELLOW, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 5, Savile row, Burlington gardens. *Trans.* 1.

1818 JAMES ALEXANDER GORDON, M.D. C.B. F.R.S., Parham House, Dorking. C. 1828. V.P. 1829. *Trans.* 1.

1851 PETER YEAMES GOWLLAND, Assistant-Surgeon to the London Hospital, and Surgeon to St. Mark's Hospital; 34, Finsbury square.

1844 JOHN GRANTHAM, Crayford, Kent.

1850 HENRY GRAY, F.R.S., Lecturer on Anatomy at St. George's Hospital Medical School, and Surgeon to the St. George's and St. James's Dispensary; 8, Wilton street, Grosvenor place. *Trans.* 2.

1846 GEORGE THOMPSON GREAM, M.D., 2, Upper Brook street, Grosvenor square.

1816 JOSEPH HENRY GREEN, D.C.L. F.R.S., Consulting Surgeon to St. Thomas's Hospital; Hadley, Middlesex. C. 1820. V.P. 1830. *Trans.* 1.

1843 ROBERT GREENHALGH, M.D., Consulting Physician-Accoucheur to the St. John's Wood Dispensary; 11, Upper Woburn place, Russell square.

1814 JOHN GROVE, M.D., Salisbury.

1852 JOHN GROVE, West Hill, Wandsworth, Surrey.

1849 WILLIAM WITHEY GULL, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital, and Member of the Senate of the University of London; 8, Finsbury square. *Trans.* 2.

1837 JAMES MANBY GULLY, M.D.; Great Malvern, Worcestershire.

1859 THEOPHILUS MILLER GUNN; 40, York place, Portman square.

1819 JOHN GUNNING, C.B., Inspector-General of Hospitals; Paris. C. 1823.

Elected

1854 SAMUEL OSBORNE HABERSHON, M.D., Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; 22, Wimpole street, Cavendish square.

1849 HAMMETT HAILEY, Newport Pagnell, Bucks.

1845 SIR JOHN HALL, M.D. K.C.B., Inspector-General of Hospitals.

1848 ALEXANDER HALLEY, M.D. F.G.S., 7, Harley street, Cavendish square.

1819 THOMAS HAMMERTON, 112, Piccadilly. C. 1829–30.

1838 HENRY HANCOCK, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital, and Surgeon to the Royal Westminster Ophthalmic Hospital; 37, Harley street, Cavendish square. C. 1851.

1849 *RICHARD JAMES HANSARD, Surgeon to the Radcliffe Infirmary; 5, Broad street, Oxford.

1848 *GEORGE HARCOURT, M.D., Chertsey, Surrey.

1836 JOHN FOSSE HARDING, Sandford House, Highbury New Park. C. 1858-9.

1856 CHARLES JOHN HARE, M.D., Assistant-Physician to University College Hospital; 41, Brook street, Grosvenor square.

1858 WILLIAM WARWICK HARKNESS, late Demonstrator of Anatomy at the London Hospital Medical College; 9, Finsbury Circus.

1857 GEORGE HARLEY, M.D. F.C.S., Professor of Medical Jurisprudence in University College, London; 77, Harley street, Cavendish square.

1859 FRANCIS HARRIS, M.B., Assistant-Physician to the Hospital for Sick Children, and Physician-Accoucheur to the St. George's and St. James's Dispensary; 11, New Cavendish street, Portland place.

1843 THOMAS SUNDERLAND HARRISON, M.D. F.L.S., Innox Hill House, Frome, Somersetshire.

1846 JOHN HARRISON, 2, the Court yard, Albany.

1841 WILLIAM HARVEY, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity, and Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.

Elected

1855 ALFRED HAVILAND, Surgeon to the Bridgewater Infirmary; Bridgewater, Somerset.

1828 CÆSAR HENRY HAWKINS, F.R.S., Surgeon Extraordinary to H.M. the Queen, and Senior Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

1838 CHARLES HAWKINS, Inspector of Anatomy, Consulting Surgeon to Queen Charlotte's Hospital; 22, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. *Trans.* 2.

1848 THOMAS HAWKSLEY, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 26, George street, Hanover square.

1820 THOMAS EMERSON HEADLAM, M.D., Consulting Physician to the Newcastle Infirmary, Newcastle-upon-Tyne.

1848 *JAMES NEWTON HEALE, M.D., Physician to the Winchester County Hospital; Winchester, Hants.

1850 GEORGE HEATON, M.D., Boston, U.S.

1829 THOMAS HEBERDEN, M.D., 43, Park street, Grosvenor square.

1844 JOHN HENNEN, M.D. L. 1848-50.

1849 AMOS HENRIQUES, M.D., Hon. Physician to the Spanish Embassy; 67, Upper Berkeley street, Portman square.

1848 MITCHELL HENRY, Surgeon to, and Lecturer on Medical Jurisprudence at, the Middlesex Hospital, and Surgeon to the North London Eye Infirmary; 5, Harley street, Cavendish square. *Trans* 2.

1821 VINCENT HERBERSKI, M.D., Professor of Medicine in the University of Wilna.

1843 PRESCOTT GARDNER HEWETT, Assistant-Surgeon to St. George's Hospital; 1, Chesterfield street, May fair. C. 1859. *Trans.* 7.

1855 GRAILY HEWITT, M.D., Physician to the British Lying-in Hospital, and to the Samaritan Free Hospital for Women and Children; Lecturer on Comparative Anatomy and Zoology at St. Mary's Hospital; 17, Radnor place, Hyde park.

Elected

1853 THOMAS HEWLETT, Surgeon to Harrow School; Harrow, Middlesex. *Trans.* 1.

1841 *NATHANIEL HIGHMORE, Sherborne, Dorsetshire.

1854 THOMAS HILLIER, M.D., Medical Officer of Health for St. Pancras, and Assistant-Physician to the Hospital for Sick Children; 21, Upper Gower street.

1842 WILLIAM AUGUSTUS HILLMAN, Senior Assistant-Surgeon to the Westminster Hospital; 1, Argyll street, Regent street, C. 1858-9.

1841 JOHN HILTON, F.R.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital, and Consulting-Surgeon to the St. Pancras Royal General Dispensary, Professor of Anatomy and Surgery at the Royal College of Surgeons; 10, New Broad Street, City. C. 1851. *Trans.* 3.

1859 FRANCIS HIRD, Visiting-Surgeon and Lecturer at the Charing Cross Hospital; 17, Clifford street, Bond street.

1848 MARTIN THOMAS HISCOX, M.D., Bath, Somersetshire.

1840 THOMAS HODGKIN, M.D., Consulting Physician to the Hospital for Diseases of the Skin, and Member of the Senate of the University of London; 35, Bedford square. C. 1842-3. *Trans.* 6.

1813 JOSEPH HODGSON, F.R.S., 60, Westbourne terrace, Hyde park gardens. C. 1817. P. 1851-2. *Trans.* 1.

1843 LUTHER HOLDEN, Lecturer on Anatomy at St. Bartholomew's Hospital, and Surgeon to the Metropolitan Dispensary; 54, Gower street, Bedford square. C. 1859.

1814 SIR HENRY HOLLAND, Bart., M.D., D.C.L., LL.D. F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince Consort; 25, Brook street, Grosvenor square. C. 1817, 1833-4. V.P. 1826, 1840. *Trans.* 1.

1856 TIMOTHY HOLMES, Curator of the Pathological Museum of St. George's Hospital, and Assistant-Surgeon to the Hospital for Sick Children; 22, Queen street, May fair. *Trans.* 1.

Elected

1846 Barnard Wight Holt, Senior Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 5, Parliament street.

1846 Carsten Holthouse, Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; Surgeon to the South London Ophthalmic Hospital; 2, Storey's gate, St. James's park.

1853 William Charles Hood, M.D., Resident Physician and Medical Superintendent of Bethlem Hospital.

1828 *Edward Howell, M.D., Senior Consulting Physician to the Swansea Infirmary; 2, South Hill place, Swansea, Glamorganshire.

1857 John Whitaker Hulke, Assistant-Surgeon to King's College Hospital, and to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington st. *Trans.* 1.

1857 Edward Charles Hulme, Assistant-Surgeon to the Central London Ophthalmic Hospital, Surgeon to the Blenheim-street Dispensary, and Examining Surgeon to the Marine Society; 19, Gower street, Bedford square.

1844 Edwin Humby, 1, Windsor terrace, Maida Hill.

1855 George Murray Humphry, F.R.S., Surgeon to, and Lecturer on Surgery at, Addenbrooke's Hospital, Cambridge. *Trans.* 2.

1840 Henry Hunt, M.R.C.P., 68, Brook street, Hanover square. C. 1857. *Trans.* 2.

1849 Edward Law Hussey, Surgeon to the Radcliffe Infirmary, St. Aldate's, Oxford. *Trans.* 1.

1856 Jonathan Hutchinson, Surgeon to the Metropolitan Free Hospital; 14, Finsbury circus. *Pro.* 2.

1820 William Hutchinson, M.D.

1840 Charles Hutton, M.D., Physician to the General Lying-in Hospital, and to the Royal Infirmary for Women and Children; 26, Lowndes street, Belgrave square. C. 1858-9.

1847 William Edmund Image, Senior Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

Elected

1856 CORNELIUS INGLIS, M.D., 2, Norfolk road, St. John's Wood.

1826 WILLIAM INGRAM, Midhurst, Sussex.

1845 *HENRY JACKSON, Senior Surgeon to the Sheffield General Infirmary; St. James's row, Sheffield, Yorkshire.

1841 PAUL JACKSON, 24, Wimpole street, Cavendish square.

1841 MAXIMILIEN MORRIS JACOBOVICS, M.D., Vienna.

1825 JOHN B. JAMES, M.D.

1847 *WILLIAM WITHALL JAMES, Surgeon to the Devon and Exeter Hospital; Exeter, Devonshire.

1844 SAMUEL JOHN JEAFFRESON, M.D., Physician to the Warneford Hospital, and Warwick Dispensary; Leamington, Warwickshire.

1839 JULIUS JEFFREYS, F.R.S., Kingston, Surrey.

1840 *GEORGE SAMUEL JENKS, M.D., 18, Circus, Bath.

1851 WILLIAM JENNER, M.D., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; Physician to the Hospital for Sick Children; 8, Harley street, Cavendish square. *Trans.* 2.

1848 ATHOL ARCHIBALD WOOD JOHNSON, Lecturer on Anatomy and Physiology at St. George's Hospital Medical School, and Surgeon to the Hospital for Sick Children; 37, Albemarle street. *Trans.* 1.

1851 EDMUND CHARLES JOHNSON, M.D., Corresponding Member of the Imperial Society of Florence, 6, Savile row.

1821 SIR EDWARD JOHNSON, M.D., Weymouth, Dorsetshire.

1847 GEORGE JOHNSON, M.D., Professor of Materia Medica and Therapeutics in King's College, London, and Physician to King's College Hospital; 11, Savile row, Burlington gardens. *Trans.* 5.

1837 HENRY CHARLES JOHNSON, Surgeon to St. George's Hospital, 6, Savile row, Burlington gardens. C. 1850-1.

1853 HENRY JONES, 36, Soho square.

1844 HENRY BENCE JONES, M.D., F.R.S., Physician to St. George's Hospital; 31, Brook street, Grosvenor square. C. 1855-6. *Trans.* 11.

1835 HENRY DERVICHE JONES, 23, Soho square. C. 1854-5.

1859 WILLIAM PRICE JONES, M.D., Surbiton, Kingston.

Elected

1853 THOMAS WHARTON JONES, F.R.S., Professor of Ophthalmic Surgery in University College, London, and Ophthalmic Surgeon to University College Hospital; 35, George street, Hanover square. *Trans.* 1.

1837 THOMAS WILLIAM JONES, M.D., Physician to the City Dispensary; 19, Finsbury pavement. C. 1858.

1829 *GEORGE CHARLES JULIUS, Richmond, Surrey.

1816 *GEORGE HERMANN KAUFFMANN, M.D., Hanover.

1848 *DANIEL BURTON KENDELL, M.D., Kettlethorpe Hall, Wakefield, Yorkshire.

1847 ALFRED KEYSER, 21, Norfolk crescent, Oxford square.

1857 HENRY WALTER KIALLMARK, late Staff Surgeon, 2d class, attached to the Ottoman Army; 46, Prince's square, Westbourne grove.

1839 *DAVID KING, M.D., Medical Officer of Health for Eltham; Eltham, Kent.

1851 JOHN ABERNETHY KINGDON, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, City.

1858 WILLIAM SENHOUSE KIRKES, M.D., Assistant-Physician to St. Bartholomew's Hospital; 2, Lower Seymour street, Portman square. *Trans.* 1.

1840 SAMUEL ARMSTRONG LANE, Surgeon to St. Mary's Hospital; and Consulting Surgeon to the Lock Hospital; 1, Grosvenor place. C. 1849-50.

1855 JAMES ROBERT LANE, Surgeon to, and Lecturer on Anatomy and Physiology at, St. Mary's Hospital; and Surgeon to the Lock and St. Mark's Hospitals; 1, Grosvenor place.

1841 *CHARLES LASHMAR, M.D., 83, North End, Croydon, Surrey.

1816 G. E. LAWRENCE.

1809 WILLIAM LAWRENCE, F.R.S., Serjeant-Surgeon to H.M. the Queen; Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital, and Surgeon to Bridewell, and Bethlem Hospital, Foreign Associate of the Imperial Academy of Medicine of Paris; 18, Whitehall place. S. 1813-7. V.P. 1818-9. T. 1821-6. P. 1831-2. C. 1820, 1833-4, 1842-3. *Trans.* 18.

Elected

1840 THOMAS LAYCOCK, M.D. F.R.S.E., Professor of the Practice of Medicine in the University of Edinburgh, and Physician to the Edinburgh Royal Infirmary; 4, Rutland street, Edinburgh.

1843 *JESSE Leach, Moss Hall, Heywood, near Bury, Lancashire.

1823 JOHN G. LEATH, M.D.

1822 JOHN JOSEPH LEDSAM, M.D., 17, Esplanade, Scarborough, Yorkshire.

1823 HENRY LEE, M.D. F.L.S., Weather Oak, Alvechurch, near Bromsgrove, Worcestershire. C. 1837. S. 1839-40.

1843 HENRY LEE, Surgeon to King's College Hospital, and Senior Surgeon to the Lock Hospital; 9, Savile row, Burlington gardens. C. 1856-7. *Trans.* 3. *Pro.* 1.

1822 ROBERT LEE, M.D. F.R.S., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; and Corresponding Member of the Imperial Academy of Medicine, Paris; 4, Savile row, Burlington gardens. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 20.

1836 FREDERICK LEIGHTON, M.D., Frankfort-on-the-Maine.

1854 HANANEL DE LEON, M.D., 6, Victoria terrace, Bedford.

1856 DAVID LEWIS, M.D., Physician to the Royal Society of Ancient Britons' Schools.

1847 SIR JOHN LIDDELL, M.D. C.B. F.R.S., Director-General of the Medical Department of the Navy; Somerset House.

1806 JOHN LIND, M.D.

1845 WILLIAM JOHN LITTLE, M.D., Physician to the London Hospital; 34, Brook street, Grosvenor square.

1819 ROBERT LLOYD, M.D.

1824 EUSEBIUS ARTHUR LLOYD, Surgeon to St. Bartholomew's and Christ's Hospitals; 14, Bedford row. S. 1827-8. V.P. 1838. C. 1843-4. *Trans.* 2.

1820 J. G. LOCHER, M.C.D., Town Physician of Zurich. *Trans.* 2.

1824 SIR CHARLES LOCOCK, Bart., M.D., First Physician-Accoucheur to H.M. the Queen, and Consulting Physician to the General Lying-in Hospital; Member of the Senate of the University of London; 26, Hertford street, Mayfair. C. 1826. V.P. 1841. P. 1857-8. *Trans.* 1.

Elected

1852 CHARLES LODGE, M.D.

1846 HENRY THOMAS LOMAX, Surgeon to the County Police; St. Mary's grove, Stafford.

1836 JOSEPH S. LÖWENFELD, M.D., Berbice.

1852 JAMES LUKE, F.R.S., Senior Surgeon to the London Hospital; and Surgeon to St. Luke's Hospital for Lunatics; 37, Broad street buildings, City. C. 1858. *Trans.* 4.

1857 FELIX WILLIAM LYON, M.D., Lyndhurst, Hants.

1846 WILLIAM M'EWEN, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

1823 GEORGE MACILWAIN, Consulting Surgeon to the Finsbury Dispensary and the St. Ann's Society's Schools; 3, the Court yard, Albany. C. 1829-30. V.P. 1848. *Trans.* 1.

1848 FREDERICK WILLIAM MACKENZIE, M.D., Physician to the Western General Dispensary; 11, Chester place, Hyde park square. *Trans.* 2.

1818 WILLIAM MACKENZIE, M.D., Surgeon-Oculist to H.M. the Queen in Scotland, and Surgeon to the Glasgow Eye Infirmary; 49, Bath street, Glasgow. *Trans.* 2.

1854 *DRAPER MACKINDER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1822 RICHARD MACINTOSH, M.D.

1844 DANIEL MACLACHLAN, M.D., Physician to the Royal Hospital, Chelsea, and Deputy Inspector-General of Hospitals; Royal Hospital, Chelsea. *Trans.* 1.

1851 SAMUEL MACLEAN, 10, Conduit street, Bond street.

1849 DUNCAN MACLACHLAN MACLURE, 16, Harley street, Cavendish square.

1842 JOHN MACNAUGHT, M.D., Bedford street, Liverpool.

1837 ANDREW MELVILLE M'WHINNIE, Assistant-Surgeon to, and Lecturer on Comparative Anatomy at, St. Bartholomew's Hospital; and Assistant-Surgeon to the London Hospital for Diseases of the Skin, Blackfriars; 5, Crescent, New Bridge street, Blackfriars. C. 1851-2. *Trans.* 1.

1855 WILLIAM MARCET, M.D. F.R.S., Assistant-Physician to, and Lecturer on Chemistry at, the Westminster Hospital; 36, Chapel street, Belgrave square. *Trans.* 1.

Elected

1848 WILLIAM ORLANDO MARKHAM, M.D., Physician to, and Lecturer on Physiology and General and Morbid Anatomy at, St. Mary's Hospital; 33, Clarges street, Piccadilly. *Trans.* 1.

1824 SIR HENRY MARSH, Bart., M.D., Physician to H.M. the Queen in Ireland, Consulting Physician to the City of Dublin Hospital, and Physician to Steevens's Hospital; 9, Merrion square north, Dublin.

1838 THOMAS PARR MARSH, M.D., Consulting Physician to the Salop Infirmary, Shrewsbury; Powyn, Merionethshire.

1851 JOHN MARSHALL, F.R.S., Assistant-Surgeon to University College Hospital; 10, Savile row, Burlington gardens. *Trans.* 2.

1841 JAMES RANALD MARTIN, F.R.S., Examining Medical Officer to the Secretary of State for India in Council; 71A, Grosvenor street. C. 1853.

1849 GEORGE BELLASIS MASPEN, Ghazeepore, India.

1853 WILLIAM EDWARD MASPEN, Surgeon to the Staffordshire General Infirmary, Stafford.

1818 J. P. MAUNOIR, Professor of Surgery at Geneva. *Trans.* 4.

1837 THOMAS MAYO, M.D. F.R.S., President of the Royal College of Physicians, and Honorary Physician to the St. Marylebone Infirmary; 56, Wimpole street, Cavendish square. S. 1841. C. 1847-8. V.P. 1851-2.

1839 RICHARD HENRY MEADE, Senior Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1837 SAMUEL WILLIAM JOHN MERRIMAN, M.D., Physician to the Royal Infirmary for Women and Children; Consulting Physician to the Westminster General Dispensary; and Physician-Accoucheur to the Western General Dispensary; 3, Charles street, Westbourne terrace. C. 1851-2. *Trans.* 1.

1852 JAMES MERRYWEATHER, 57, Brook street, Grosvenor square.

1847 EDWARD MERYON, M.D., *Librarian*; 14, Clarges street, Piccadilly. *Trans.* 1.

1815 AUGUSTUS MEYER, M.D., St. Petersburgh.

1840 RICHARD MIDDLEMORE, Consulting Surgeon to the Birmingham Eye Infirmary; Temple row, Birmingham.

Elected

1854 EDWARD ARCHIBALD MIDDLESHIP, late of Richmond, Surrey.

1818 *PATRICK MILLER, M.D. F.R.S. Edinb., Senior Physician to, the Devon and Exeter Hospital, and to St. Thomas's Hospital for Lunatics; the Grove, Exeter, Devonshire.

1844 NATHANIEL MONTEFIORE, 36, Hyde park gardens.

1848 CHARLES HEWITT MOORE, *Secretary*, Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 102, Piccadilly. L. 1858. *Trans.* 3.

1836 GEORGE MOORE, M.D., Hastings, Sussex.

1857 JOHN MORGAN, 3, Sussex place, Hyde park gardens.

1854 GEORGE MOSELEY (late of Sandgate).

1851 FREDERICK JOHN MOUAT, M.D., Professor of Medicine in the Medical College of Calcutta, and Secretary of the Council of Education in India; Calcutta.

1856 CHARLES MURCHISON, M.D., Assistant-Physician to King's College Hospital, and to the London Fever Hospital; 79, Wimpole street, Cavendish square. *Trans.* 2.

1847 SIMON MURCHISON, Bicester, Oxon.

1859 GEORGE NAYLER, Assistant-Surgeon, Bombay Army; Jhansi, Central India, and Junior United Service Club.

1835 THOMAS ANDREW NELSON, M.D., 10, Nottingham terrace, York gate, Regent's park [54, Marylebone road].

1843 EDWARD NEWTON, 30, Fitzroy square.

1851 JAMES NICHOLS, 13, Savile row, Burlington gardens.

1819 *GEORGE NORMAN, Consulting Surgeon to the Bath United Hospital, and Surgeon to the Puerperal Charity; Circus, Bath. *Trans.* 3.

1849 HENRY BURFORD NORMAN, Portland Lodge, Southsea, Hants.

1845 HENRY NORRIS, Charmouth, Dorset.

1847 *WILLIAM EDWARD CHARLES NOURSE, 11, Old Steine, Brighton.

1849 *ARTHUR NOVERRE, Great Stanmore, Middlesex.

1859 *THOMAS NUNNELEY, Senior Surgeon to the Leeds Eye and Ear Infirmary; Leeds. *Trans.* 1.

1847 THOMAS O'CONNOR, March, Cambridgeshire.

1843 WILLIAM O'CONNOR, M.D., Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

Elected

1846 FRANCIS ODLING, 52, Devonshire street, Portland place.

1858 WILLIAM MACKAY OGILVIE, Boughton Blean, near Faversham, Kent.

1858 JOHN WILLIAM OGLE, M.D., Assistant-Physician to St. George's Hospital; 13, Upper Brook street, Grosvenor square. *Trans.* 2.

1855 WILLIAM OGLE, M.A., M.D., Physician to the Royal Pimlico Dispensary; 9, Lower Belgrave street, Eaton square.

1850 HENRY OLDHAM, M.D., Obstetric Physician to, and Clinical Lecturer on Midwifery at, Guy's Hospital; and Obstetric Physician to the Tower Hamlets Dispensary; 26, Finsbury square. *Trans.* 1.

1842. WILLIAM PIERS ORMEROD.

1846 *EDWARD LATHAM ORMEROD, M.D., Physician to the Sussex County Hospital; 14, Old Steine, Brighton. *Trans.* 2.

1847 *WILLIAM BOUSFIELD PAGE, Surgeon to the Cumberland Infirmary; Carlisle. *Trans.* 2.

1840 JAMES PAGET, F.R.S., Surgeon Extraordinary to H.M. the Queen; Assistant-Surgeon to St. Bartholomew's Hospital; 1, Harewood place, Hanover square, C. 1848-9. *Trans.* 8.

1858 *WILLIAM PALEY, M.D., Physician to the Halifax Infirmary; Carlton place, Halifax, Yorkshire.

1836 S. W. LANGSTON PARKER, Surgeon to the Queen's Hospital, Birmingham; Colmore row, Birmingham.

1847 NICHOLAS PARKER, M.D., Assistant-Physician to, and Lecturer on Medicine at, the London Hospital; 22, Finsbury square.

1841 JOHN PARKIN, M.D., Rome.

1851 JAMES PART, 7, Camden road villas, Camden town.

1828 RICHARD PARTRIDGE, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; 17, New street, Spring gardens. S. 1832-6, C. 1837-8. V.P. 1847-8.

Elected

1845 THOMAS BEVILL PEACOCK, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, St. Thomas's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria park; 20, Finsbury circus. S. 1855-6. *Trans.* 2.

1856 RICHARD KING PEIRCE, Surgeon in Diseases of Women and Children to the Blenheim street Dispensary; 16, Norland place, Notting hill.

1830 CHARLES P. PELECHIN, M.D., St. Petersburgh.

1855 *OLIVER PEMBERTON, Surgeon to, and Lecturer on Clinical Surgery at, the Birmingham General Hospital, 18, Temple row, Birmingham.

1844 WILLIAM VESALIUS PETTIGREW, M.D., Surgeon to the Female Orphan Asylum, Lambeth; 7, Chester street, Grosvenor place.

1837 BENJAMIN PHILLIPS, F.R.S., Brentbridge House, Hendon, Middlesex. L. 1841-5. T. 1847-50. V.P. 1853. *Trans.* 4.

1848 EDWARD PHILLIPS, M.D. F.L.S., Physician to the Coventry and Warwickshire Hospital; Coventry, Warwickshire.

1852 RICHARD PHILLIPS, Winchester place, Claremont square, Pentonville [68, Pentonville road].

1854 THOMAS BACON PHILLIPS, 36, Lansdown place, Brighton.

1846 FRANCIS RICHARD PHILP, M.D.

1851 *JAMES HOLLINS PICKFORD, M.D. M.R.I.A., 1, Cavendish place, Brighton.

1851 JOHN PICTON, M.D.

1836 ISAAC PIDDUCK, M.D., Physician to the Bloomsbury Dispensary; 22, Montague street, Russell square. *Pro.* 2.

1852 HENRY PILLEAU, Staff Surgeon, 1st Class; 21, Kensington square. (India.)

1841 HENRY ALFRED PITMAN, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 94, Gloucester place, Portman square. L. 1851-3.

1850 ALFRED POLAND, Assistant-Surgeon to Guy's Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Bolton row, May fair.

Elected

1845 George David Pollock, F.L.S., *Librarian*, Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 27, Grosvenor street. C. 1856-7. *Trans.* 2.

1843 Charles Pope, M.D. F.L.S., Glastonbury, Somersetshire.

1846 Jephson Potter, M.D. F.L.S., Sussex House, Pittville, Cheltenham.

1842 James Powell, M.B., 77, Guildford street, Russell square.

1851 Robert Francis Power, M.D., 7, Lower Grosvenor place.

1857 William Overend Priestley, M.D., Physician-Accoucheur to the St. Marylebone Infirmary, and to the St. George's and St. James's Dispensary; Physician to the Samaritan Free Hospital; 31, Somerset street, Portman square.

1839 John Propert, Consulting-Surgeon to the Society of Ancient Britons; 6, New Cavendish street, Portland place.

1845 John Pyle, 56, Oxford terrace, Hyde park.

1816 Sir William Pym, M.D., K.C.H., Inspector-General of Army Hospitals, and Superintendent-General of Quarantine in the United Kingdom.

1830 Jones Quain, M.D., Paris.

1850 Richard Quain, M.D,, Physician to the Hospital for Consumption and Diseases of the Chest; 23, Harley street, Cavendish square. *Trans.* 1.

1853 Richard Quain, F.R.S., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

1852 Charles Bland Radcliffe, M.D., Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; 4, Henrietta street, Cavendish square.

1857 Henry Ranke, M.D. (Germany).

1854 William Henry Ransom, M.D., Physician to the Nottingham General Hospital; Nottingham.

1858 Frederick George Reed, M.D., 46, Hertford st., May fair.

1821 Henry Reeder, M.D.

Elected

1857 George Owen Rees, M.D. F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Examiner in Materia Medica at the University of London; 26, Albemarle street, Piccadilly. *Trans.* 1.

1835 G. Regnoli, Professor of Surgery in the University of Pisa.

1855 John Russell Reynolds, M.D., Assistant-Physician to the Westminster Hospital; 38, Grosvenor street.

1847 Samuel Richards, M.D., 36, Bedford square.

1852 Christopher Thomas Richardson, M.B., Physician to the Metropolitan Free Hospital; 16, Hinde street, Manchester square.

1829 Sir John Richardson, Knt., M.D. LL.D. C.B. F.R.S., Inspector of Hospitals and Fleets; Lancrigg, Grasmere, Westmoreland.

1849 *William Richardson, M.D., 9, Ephraim road, Tunbridge Wells, Kent.

1845 Benjamin Ridge, M.D., 21, Bruton street, Berkeley square.

1843 Joseph Ridge, M.D., 39, Dorset square. C. 1858. *Pro.* 1.

1852 Charles Ridley, Surgeon to the Royal Society for Protection of Life from Fire; 6, Charlotte street, Bedford sq.

1852 John Roberts, M.R.C.P., 75, Grosvenor street.

1829 *Archibald Robertson, M.D. F.R.S., Hon. Physician to the Northampton General Infirmary, Northampton.

1855 Charles Alexander Lockhart Robertson, M.D., Medical Superintendent of the Sussex County Lunatic Asylum, and Hon. Secretary to the Association of Medical Officers of Asylums and Hospitals for the Insane; Hayward's Heath, Sussex.

1857 John George Robertson, Assistant Medical Officer, County Lunatic Asylum; Exminster, Devonshire.

1843 George Robinson, M.D., Physician to the Newcastle-upon-Tyne Dispensary; Eldon square, Newcastle-upon-Tyne. *Trans.* 2.

1843 William Roden, M.D. F.L.S., the Grange, Kidderminster, Worcestershire.

1835 George Hamilton Roe, M.D., Senior Physician to the Hospital for Consumption and Diseases of the Chest; 57, Park street, Grosvenor square. C. 1841-2. *Trans.* 1.

Elected

1836 ARNOLD ROGERS, Consulting Surgeon-Dentist to St. Bartholomew's Hospital; 16, Hanover square.

1846 WILLIAM RICHARD ROGERS, M.D., Physician to the Farringdon General Dispensary and Lying-in Charity, and to the Western General Lying-in Institution; 56 Berners street.

1819 HENRY SHUCKBURGH ROOTS, M.D., Consulting Physician to St. Thomas's Hospital; 2, Russell square. C. 1833, 1845. V.P., 1834-5. *Trans.* 1.

1829 WILLIAM SUDLOW ROOTS, F.L.S., Surgeon to the Royal Establishment at Hampton Court, and to the Kingston Dispensary; Kingston, Surrey.

1850 GEORGE ROPER, 180, Shoreditch.

1836 RICHARD ROSCOE, M.D.

1855 THOMAS TATTERSALL ROSCOW, M.D., Physician to the Chelsea, Brompton, and Belgrave Dispensary; 1, Sumner place, Onslow square, Brompton.

1836 *CALEB BURRELL ROSE, F.G.S., 25, King street, Great Yarmouth, Norfolk. *Trans.* 1.

1857 HENRY COOPER ROSE, M.D., High street, Hampstead.

1849 CHARLES HENRY FELIX ROUTH, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.

1845 HENRY MORTIMER ROWDON, Member of the Court of Examiners of the Society of Apothecaries; 29, Nottingham place, Marylebone road.

1834 HENRY WYLDBORE RUMSEY, Gloucester lodge, Cheltenham.

1845 JAMES RUSSELL, M.D., Lecturer on Pathology and Therapeutics at Sydenham College; 91, New Hall street, Birmingham.

1851 HENRY HYDE SALTER, M.D., F.R.S., Assistant-Physician to, and Lecturer on Physiology and Pathology at, the Charing Cross Hospital; 6, Montague street, Russell square.

1856 SAMUEL JAMES A. SALTER, F.L.S, Surgeon-Dentist to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. *Trans.* 1.

1849 HUGH JAMES SANDERSON, M.D., Physician to the Hospital for Women; 26, Upper Berkeley street, Portman square.

Elected

1855 JOHN BURDON SANDERSON, M.D., Medical Officer of Health for Paddington; Lecturer on Medical Jurisprudence at St. Mary's Hospital; 9, Gloucester place, Hyde park.

1847 WILLIAM HENRY OCTAVIUS SANKEY, M.D., Middlesex County Lunatic Asylum, Hanwell.

1845 EDWIN SAUNDERS, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince Consort; 13A, George street, Hanover square.

1834 LUDWIG V. SAUVAN, M.D., Warsaw.

1859 WILLIAM SCOVELL SAVORY, Lecturer on General Anatomy and Physiology at St. Bartholomew's Hospital, and Surgeon to the Great Northern Hospital; Professor of Comparative Anatomy and Physiology at the Royal College of Surgeons; 13, Charterhouse square. *Trans.* 2.

1840 AUGUSTIN SAYER, M.D., Consulting Physician to the Lock Hospital; 28, Upper Seymour street, Portman square.

1853 MAURICE SCHULHOF, M.D., Physician to the Royal General Dispensary, Bartholomew close; 14, Brook street, Grosvenor square.

1858 *GEORGE SCRATCHLEY, M.D., New Orleans, Louisiana, U.S.

1856 EDWIN SERCOMBE, Surgeon-Dentist to St. Mary's Hospital; 49, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1824 EDWARD JAMES SEYMOUR, M.D. F.R.S., Consulting Physician to the Seamen's Hospital Ship 'Dreadnought;' 13, Charles street, Berkeley square. C. 1826, 1831. S. 1827-8. V.P. 1830, 1842. *Trans.* 2.

1840 WILLIAM SHARP, M.D. F.R.S., Horton House, Rugby. *Trans.* 1.

1837 WILLIAM SHARPEY, M.D. F.R.S., Examiner in Anatomy at the University of London, Professor of Anatomy and Physiology in University College, London, and Secretary of the Royal Society; 33, Woburn place, Russell square. C. 1848-9.

1836 ALEXANDER SHAW, *Treasurer*, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 22A, Cavendish square. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858. *Trans.* 4.

Elected

1848 *Edward James Shearman, M.D., Rotherham, Yorkshire.

1859 Septimus William Sibley, Lecturer on Pathological Anatomy at the Middlesex Hospital; 12, New Burlington street. *Trans.* 2.

1849 Francis Sibson, M.D. F.R.S., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 40, Brook street, Grosvenor square. *Trans.* 1.

1848 Edward Henry Sieveking, M.D., Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; 17, Manchester square. C. 1859. *Trans.* 1.

1839 Thomas Hookham Silvester, M.D., Medical Officer to the Clapham General Dispensary; High street, Clapham. C. 1854-5. *Trans.* 1.

1842 John Simon, F.R.S., Surgeon to, and Lecturer on Pathology at, St. Thomas's Hospital; Medical Officer of the Privy Council; 44, Cumberland street, Bryanston square. C. 1854-55. *Trans.* 1.

1857 James Lewis Siordet, M.B., late Physician to the Blenheim Free Dispensary and Infirmary.

1827 George Robert Skene, Bedford.

1824 Frederic Carpenter Skey, F.R.S., *President*, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 13, Grosvenor street. C. 1828. L. 1829-31. V.P. 1841-2. *Trans.* 1.

1852 Charles Case Smith, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.

1854 Edward Smith, M.D. LL.B., Assistant-Physician to the Hospital for Consumption and Diseases of the Chest; 6, Queen Anne street, Cavendish square. *Trans.* 5.

1835 John Gregory Smith, Harewood, Leeds, Yorkshire.

1843 Robert William Smith, M.D. M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; 63, Eccles street, Dublin.

1838 Spencer Smith, *Vice-President*, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 48, Sussex gardens, Hyde park. C. 1854. S. 1855-8.

1845 William Smith, Chesterfield, Derbyshire.

Elected

1847 WILLIAM SMITH, M.D., Consulting Physician to the Weymouth Infirmary, Weymouth, Dorsetshire.

1850 WILLIAM TYLER SMITH, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 7, Upper Grosvenor street. *Trans.* 2.

1851 JOHN SODEN, Surgeon to the Bath United Hospital, and Consulting Surgeon to the Bath Eye Infirmary; 24, Circus, Bath. *Trans.* 2.

1816 *JOHN SMITH SODEN, 101, Sidney place, Bath. *Trans.* 1.

1830 SAMUEL SOLLY, F.R.S., Surgeon to St. Thomas's Hospital, and Consulting Surgeon to the Royal General Dispensary, Bartholomew close; 18, St. Helen's place, Bishopsgate street. L. 1838-40. C. 1845-6. V.P. 1849-50. *Trans.* 6.

1844 FREDERICK ROBERT SPACKMAN, M.D., Harpenden, St. Alban's.

1834 JAMES SPARK, Italy.

1851 ROBERT JOHN SPITTA, M.B., Medical Officer to the Clapham General Dispensary; Clapham, Surrey. *Trans.* 1.

1843 *STEPHEN SPRANGER, Hursley, Hampshire.

1858 JOSHUA HARRISON STALLARD, M.B. Lond., Physician to the St. George's and St. James's Dispensary; 12, Welbeck street, Cavendish square.

1815 EDWARD STANLEY, F.R.S., Surgeon Extraordinary to H.M. the Queen; Surgeon to St. Bartholomew's Hospital; 23A, Brook street, Grosvenor square. C. 1821-2, 1835, 1845-6, 1852-3. S. 1824. V.P. 1827, 1839-40. T. 1832-4. P. 1843-4 *Trans.* 12.

1857 JOHN STANTON, M.D., 7, Upper George street, Bryanston square.

1851 JAMES STARTIN, Surgeon to, and Lecturer on Cutaneous Disorders at, the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.

1858 EDWARD STEPHENS, M.D., Consulting Surgeon to the Manchester Lying-in Hospital; 58, Bridge street, Manchester.

1854 HENRY STEVENS, M.B., Resident Medical Officer, St. Luke's Hospital for Lunatics, Old street.

Elected

1842 ALEXANDER PATRICK STEWART, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 74, Grosvenor street. C. 1856-7.

1859 WILLIAM EDWARD STEWART, 8, Weymouth street, Portland place.

1856 ALONZO HENRY STOCKER, M.D., Resident Medical Superintendent of Grove Hall Lunatic Asylum, Bow.

1843 ROBERT REEVE STORKS, Paris.

1858 JOHN FREMLYN STREATFEILD, Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 15, Upper Brook street, Grosvenor square.

1839 ALEXANDER JOHN SUTHERLAND, M.D. F.R.S., *Vice-President*, Senior Physician to St. Luke's Hospital for Lunatics, and Consulting Physician to the Royal South London Dispensary; 6, Richmond terrace, Whitehall. C. 1850-1. V.P. 1858. *Trans.* 1.

1855 JOHN MAULE SUTTON, M.D., Kent House, Tenby, South Wales.

1842 JAMES SYME, F.R.S.Edinb., Professor of Clinical Surgery in the University of Edinburgh, and Surgeon to the Edinburgh Royal Infirmary; 2, Rutland street, Edinburgh. *Trans.* 3.

1854 *FREDERICK SYMONDS, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 32, Beaumont street, Oxford.

1844 RICHARD WILLIAM TAMPLIN, Surgeon to the Royal Orthopædic Hospital; 33, Old Burlington street.

1848 THOMAS HAWKES TANNER, M.D. F.L.S., 10, Charlotte street, Bedford square.

1852 ROBERT TAYLOR, Surgeon to the Central London Ophthalmic Hospital, and to the Cripple's Home, Hill street; 10, George street, Hanover square.

1845 THOMAS TAYLOR, Lecturer on Chemistry at the Middlesex Hospital Medical School; 4, Vere street, Cavendish square.

1856 THOMAS PRIDGIN TEALE, F.L.S., Surgeon to the Leeds General Infirmary; 22, Albion street, Leeds. *Trans.* 2.

Elected

1859 EDWARD TEGART, Junior, 49, Jermyn street, St. James's.

1845 *EVAN THOMAS, Cheetham hill road, Manchester.

1857 HENRY THOMPSON, M.D., Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital; 75, Harley street, Cavendish square.

1852 HENRY THOMPSON, Surgeon to the St. Marylebone Infirmary, and Assistant-Surgeon to University College Hospital; 16, Wimpole street, Cavendish square. *Trans.* 2.

1839 SETH THOMPSON, M.D., 16, Lower Berkeley street, Portman square. C. 1849. S. 1850-1. V.P. 1857.

1842 THEOPHILUS THOMPSON, M.D. F.R.S., 3, Bedford square. C. 1855-6. *Trans.* 5.

1835 FREDERICK HALE THOMSON, Consulting Surgeon to the Westminster Hospital, and to the West London Institution for Diseases of the Eye; 4, Clarges street, Piccadilly.

1819 JOHN THOMSON, M.D. F.L.S., Senior Physician to the Finsbury Dispensary; 18, Dalby terrace, Islington, [364, City Road]. C. 1833. L. 1834-7. V.P. 1850-1.

1850 ROBERT DUNDAS THOMSON, M.D. F.R.S., Lecturer on Chemistry at St. Thomas's Hospital, Examiner in Chemistry at the University of London, and Medical Officer of Health for St. Marylebone; 41, York terrace, Regent's park. *Trans.* 2.

1836 JOHN THURNAM, M.D., Resident Medical Superintendent of the Wilts County Asylum, Devizes, Wiltshire. *Trans.* 4.

1848 EDWARD JOHN TILT, M.D., Physician to the Farringdon General Dispensary and Lying-in Charity; 11, York street, Portman square.

1834 ROBERT BENTLEY TODD, M.D. F.R.S., Physician to King's College Hospital; 26, Brook street, Grosvenor square. L. 1842-6. T. 1850-1. V.P. 1854. *Trans.* 3.

1828 JAMES TORRIE, M.D., Old Bridge of Don, by Aberdeen.

1843 JOSEPH TOYNBEE, F.R.S., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, Consulting Aural Surgeon to the Asylum for the Deaf and Dumb, and to the St. George's and St. James's Dispensary; 18, Savile row, Burlington gardens. *Trans.* 6. *Pro.* 1.

Elected

1850 SAMUEL JOHN TRACY, Surgeon-Dentist to St. Bartholomew's and Christ's Hospitals; 28, Old Burlington street.

1859 EDWIN THOMAS TRUMAN, 23, Old Burlington street.

1855 JAMES STEWART TULLOCH, M.D., 33, Pembridge villas, Bayswater.

1835 JOHN CUSSON TURNER, M.D., Bexley Heath, Kent.

1845 THOMAS TURNER, F.L.S., Consulting Surgeon to the Manchester Royal Infirmary, and Lecturer on Anatomy and Physiology at the Manchester Royal School of Medicine; 77, Mosley street, Manchester.

1846 ALEXANDER URE, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Westminster General Dispensary; 18, Upper Seymour street, Portman square. *Trans.* 1.

1819 BARNARD VAN OVEN, M.D., Consulting Surgeon to the Charity for Delivering Jewish Lying-in Women; 22, Manchester square.

1806 BOYER VAUX, M.D.

1810 JAMES VOSE. *Trans.* 1.

1828 BENEDETTO VULPES, M.D., Physician to the Hospital of Aversa, and to the Hospital of Incurables, Naples.

1854 EDWARD WADDINGTON, Surgeon to the King's Own Staffordshire Rifles; Wakefield, Yorkshire.

1841 ROBERT WADE, Senior Surgeon to the Westminster General Dispensary; 68, Dean street, Soho. *Trans.* 1.

1823 WILLIAM WAGNER, M.D., Berlin.

1820 THOMAS WALKER, M.D., Physician to the Forces; Morro Velhio, Brazil.

1852 WALTER HAYLE WALSHE, M.D., Professor of the Theory and Practice of Medicine in University College, London, and Physician to University College Hospital; Consulting Physician to the Hospital for Consumption; 40, Queen Anne street, Cavendish square. *Trans.* 1.

1851 HENRY HAYNES WALTON, Surgeon to the Central London Ophthalmic Hospital, and Surgeon to St. Mary's Hospital; 69, Brook street, Hanover square. *Trans.* 1. *Pro.* 1.

Elected

1852 DANIEL WANE, M.D., Obstetric Physician to the Blenheim-street Dispensary; 20, Grafton street, Berkeley square.

1846 NATHANIEL WARD, Assistant-Surgeon to the London Hospital; 1, Broad-street buildings, City. C. 1857. *Trans.* 1.

1821 WILLIAM TILLEARD WARD, Duncannon House, Brighton.

1858 JOHN RICHARD WARDELL, M.D., 4, Belmont, Tunbridge Wells.

1846 JAMES THOMAS WARE, Consulting Surgeon to the Finsbury Dispensary, and Hon. Surgeon to the Metropolitan Convalescent Institution; 51, Russell square.

1818 JOHN WARE, Clifton, near Bristol.

1814 MARTIN WARE, 51, Russell square. C. 1844–5. T. 1846. V.P. 1853.

1829 ELIAS TAYLOR WARRY, M.D., Yeovil, Somerset.

1837 THOMAS WATSON, M.D. F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840–1, 1852. V.P. 1845–6.

1847 *THOMAS WATSON, Holbeach, Lincolnshire.

1854 WILLIAM WEBB, M.D., Wirksworth, Derbyshire.

1840 WILLIAM WOODHAM WEBB, M.D., Lowestoft, Suffolk.

1842 FREDERIC WEBER, M.D., Assistant-Physician to the Middlesex Hospital, 44, Green street, Park lane. C. 1857.

1857 HERMANN WEBER, M.D., Physician to the German Hospital; 49, Finsbury square.

1835 JOHN WEBSTER, M.D. F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 24, Brook street, Grosvenor square. C. 1843-4. V.P. 1855-6. *Trans.* 5.

1844 WILLIAM WEGG, M.D., Physician to the St. George's and St. James's Dispensary; 49, Maddox street, Hanover square. L. 1854-8.

1854 THOMAS SPENCER WELLS, Lecturer on Surgery at the Grosvenor-place School of Anatomy and Medicine, and Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. *Trans.* 1.

Elected

1816 Sir Augustus West, Knt., M.D., Deputy-Inspector of Army Hospitals to the Portuguese Forces; Paris.

1842 Charles West, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; and Physician to the Hospital for Sick Children; 61, Wimpole street, Cavendish square. C. 1855-6. *Trans.* 2.

1841 Thomas West, M.D., Daventry, Northamptonshire.

1828 John Whatley, M.D.

1849 John White.

1852 John Wiblin, Medical Inspector of Emigrants and Recruits; 73, Morland place, Southampton.

1824 *William John Wickham, Consulting Surgeon to the Hants County Hospital; Winchester, Hants. *Trans.* 1.

1844 Frederic Wildbore, 1, Trafalgar place east, Hackney road.

1837 George Augustus Frederick Wilks, M.D.

1840 Charles James Blasius Williams, M.D. F.R.S., Consulting Physician to the Hospital for Consumption; 49, Upper Brook street, Grosvenor square. C. 1849-50.

1829 Robert Willis, M.D., Barnes, Surrey. L. 1838-41.

1839 Erasmus Wilson, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; 17, Henrietta street, Cavendish square. *Trans.* 2.

1839 James Arthur Wilson, M.D., *Vice-President;* 28, Dover street, Piccadilly. C. 1846-7. V.P. 1858. *Trans.* 4.

1850 *Robert Stanton Wise, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 Thomas Alexander Wise, M.D. F.R.S.E.; 17, Abercrombie place, Edinburgh.

1841 George Leighton Wood, Surgeon to the Bath General Hospital; 27, Queen square, Bath.

1851 John Wood, Assistant-Surgeon to King's College Hospital; 4, Montague street, Russell square.

1848 William Wood, M.D., 54, Upper Harley street.

1843 John Ward Woodfall, M.D., Physician to the West Kent Infirmary; Maidstone, Kent.

Elected

1833 THOMAS WORMALD, Assistant-Surgeon to St. Bartholomew's Hospital, and Surgeon to the Foundling Hospital; 42, Bedford row. C. 1839. V.P. 1854.

1842 WILLIAM COLLINS WORTHINGTON, Senior Surgeon to the Lowestoft Infirmary; Lowestoft, Suffolk. *Trans.* 3.

1848 EDWARD JOHN WRIGHT, 13, Montague place, Clapham road.

1855 HENRY G. WRIGHT, M.D., Physician to the St. Pancras Royal General Dispensary; 23, Somerset street, Portman square.

[It is particularly requested, that any change of Title, Appointment, or Residence may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1841 WILLIAM THOMAS BRANDE, D.C.L. F.R.S., Hon. Professor of Chemistry at the Royal Institution of Great Britain, Examiner in Chemistry, and Member of the Senate of the University of London; Royal Mint, Tower hill.

1835 SIR DAVID BREWSTER, K.H. D.C.L. LL.D. F.R.S., &c., Corresp. Memb. Institute of France, Principal of the University of Edinburgh.

1853 BENJAMIN COLLINS BRODIE, B.A. F.R.S., Aldrichian Professor of Chemistry in the University of Oxford.

1847 EDWIN CHADWICK, late Commissioner of the Board of Health.

1835 MICHAEL FARADAY, D.C.L. F.R.S., Corresp. Memb. Institute of France, Member of the Senate of the University of London, and Fullerian Professor of Chemistry in the Royal Institution.

1857 WILLIAM FARR, M.D. D.C.L. F.R.S., General Register Office, Somerset House.

1841 SIR JOHN FREDERICK WILLIAM HERSCHEL, Bart., D.C.L. F.R.S., Corresp. Memb. Institute of France; Collingwood, near Hawkhurst, Kent.

1835 SIR WILLIAM JACKSON HOOKER, K.H. D.C.L. LL.D. F.R.S. F.L.S., Director of the Royal Botanic Gardens, Kew; West Park, Kew.

1847 RICHARD OWEN, D.C.L. LL.D. F.R.S., Corresp. Memb. Institute of France (Foreign Associate of the Academy of Sciences); Superintendent of the Natural History Departments in the British Museum; Sheen Lodge, Mortlake.

1859 JOHN THOMAS QUEKETT, F.L.S., Conservator of the Museum, and Professor of Histology at the Royal College of Surgeons of England.

1835 The Rev. ADAM SEDGWICK, A.M. F.R.S, &c., late Woodwardian Professor of Geology, Cambridge.

1841 The Rev. WILLIAM WHEWELL, D.D. F.R.S., Master of Trinity College, Cambridge.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 G. ANDRAL, M.D., Member of the Institute and of the Imperial Academy of Medicine, Physician in Ordinary to the Emperor of the French, Professor of Pathology in the Faculty of Medicine; Paris.

1856 BARON PAUL DUBOIS, Commander of the Legion of Honour, Member of the Imperial Academy of Medicine, Dean of, and Professor of Clinical Midwifery in, the Faculty of Medicine; Paris.

1835 CARL JOHAN EKSTRÖMER, M.D. C.M. K.P.S. and W., Physician to the King of Sweden, President of the College of Health, and Director General of Hospitals; Stockholm.

1841 CHRISTIAN GOTTFRIED EHRENBERG, Berlin.

1859 J. HENLE, M.D., Professor of Anatomy at Göttingen.

1841 JAMES JACKSON, M.D. LL.D., Emeritus Professor of Medicine in the University of Cambridge, Boston, U.S.

1856 BERNHARD LANGENBECK, M.D., Professor of Surgery in the University of Berlin.

1843 BARON JUSTUS VON LIEBIG, M.D., Professor of Chemistry in the University of Munich.

1841 P. C. A. LOUIS, M,D., Honorary Physician to the Hôtel-Dieu, Member of the Imperial Academy of Medicine; Paris.

1847 CARLO MATTEUCCI, Professor in the University of Pisa, Member of the Institute of France.

1853 VALENTINE MOTT, M.D. LL.D., Emeritus Professor of Surgery in the University of New York, late President of the New York Academy of Medicine; New York.

1841 BARTOLOMEO PANIZZA, M.D., Pavia.

Elected

1859 PIERRE RAYER, M.D., late Physician to the "Hôpital de la Charité," Commander of the Legion of Honour, Member of the Institute, and of the Imperial Academy of Medicine; Paris.

1850 CARL ROKITANSKY, M.D., Curator of the Imperial Pathological Museum, and Professor at the University of Vienna.

1856 LOUIS STROMEYER, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.

1835 FRIEDRICH TIEDEMANN, M.D., Frankfort-on-the Maine.

1856 A. VELPEAU, Member of the Institute, and of the Imperial Academy of Medicine, Professor in the Faculty of Medicine, Surgeon to the "Hôpital de la Charité;" Paris.

1856 RUDOLPH VIRCHOW, M.D., Professor of Pathological Anatomy in the University of Berlin.

1859 W. VROLIK, M.D., Professor of Natural History at Amsterdam.

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A CASE OF EXCISION

OF THE

HEAD OF THE HUMERUS

(DECAPITATIO HUMERI);

WITH ITS RESULTS.

BY

JOHN BIRKETT,

SURGEON TO GUY'S HOSPITAL; FELLOW OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND, OF THE SOCIÉTÉ DE CHIRURGIE OF PARIS, AND OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON.

Received Nov. 8th.—Read Dec. 14th, 1858.

THOMAS B—, æt. 57, residing at Monckton, in the Isle of Thanet, and pursuing the occupation of a bailiff, was admitted into Guy's Hospital, July 10th, 1855, under my care.

He was emaciated and cachectic; but, until after the receipt of an injury which gave rise to his present condition, he had enjoyed robust health. Two years and five months before I saw him, a cow ran against his left leg, and thrust his right shoulder, violently, against a stone wall. The right clavicle was broken, and the shoulder was much contused. Inflammation of the shoulder-joint ensued, which terminated in suppuration. Three abscesses pointed, which were opened by Mr. Freeman, the surgeon

of Minster, under whose care the man had been from the moment of the injury. The whole of this period was passed in great suffering, and his health had declined in consequence.

When admitted, the condition of the right upper extremity was as follows:—The arm and forearm were more or less œdematous; the least movement of the member gave him intense pain, and pus escaped from three sinuses behind, and on the outer region of the shoulder. This part was swollen, but there was not any deformity. Distinct "crepitus" was felt on moving the arm, and the head of the humerus seemed to glide away, and to be removed from its normal relations with the glenoid cavity.

Believing that it was not possible to cure the disease, which probably existed in either the head of the humerus or the glenoid cavity, or both, I suggested to the man the removal of the diseased bone, and to this proposal he readily assented.

The operation was performed on July 24th, 1855. I made a triangular flap of the integuments and deltoid muscle, with its base above, and reflected it upwards. No difficulty was experienced in reaching the head of the humerus, or in exposing it to view. Sawing off the head of the bone was also easily performed with a fine "bow-saw." A rather profuse hæmorrhage delayed the operation; the blood flowed from the posterior circumflex artery, which had not been completely divided. The man was fully under the influence of chloroform during the operation.

The articular cartilage of the head of the humerus was entirely destroyed, and, at one point, a small piece of necrosed bone was lodged in an excavation, lined with granulations, close to the articular surface. The sawn surface of the bone seemed to be quite healthy, as well as the bone forming the glenoid cavity of the scapula, the articular cartilage being destroyed. There was a large quantity of the usual vascular granulations within the capsule into which the sinuses led. After maceration, the articular osseous lamella covering the cancellous tissue of the caput

humeri showed small foramina, just as if worm-eaten; but, in other respects, it was hard and smooth in some parts, perhaps from attrition. The focus of the disease appeared to be in the cancellous tissue situated between the great tuberosity of the shaft and the articular head; and here was an excavation, in which the piece of necrosed bone, before mentioned, was lodged. This dead piece of bone originally formed a small part of the superior and posterior circumference of the head, and the corresponding portion of the cervix of the bone. Upon it may be seen a part of the groove generally so distinctly marked in this spot.

Sutures were employed to adapt the edges of the wound, which quickly healed; nutritious food and stimuli were given freely, and the man left the hospital, August 18th, 1855, twenty-five days after the operation. At this date, the pain by which he had been tortured had ceased, his health improved immediately afterwards, and these were the first good results obtained.

October 30th, 1855.—The sinuses formed in the line of the cicatrix were not yet healed; but a thin, purulent discharge escaped from them. The general health of the patient was much improved, as well as his constitutional nutrition.

July, 1856.—A year after the operation. Pus still continued to flow from three sinuses, but, with a long probe, there was not any exposed bone to be detected. His general health was very good. Injections of the compound tincture of iodine were now used along the sinuses, and at the end of 1856 they had quite healed.

During the whole of this time the upper extremity had been well supported in a sling; but the man had used his forearm and hand, although to a limited degree.

September, 1858.—Since the end of 1856 this man has used his arm freely in the varied occupations of a farm-labourer. He can do any work which does not require a greater elevation of the arm than at a right angle with the body. The deltoid muscle has not sufficient power to raise it above the level of the acromion process of the scapula,

nor has the upper end of the humerus obtained a sufficiently firm and fixed point upon which to move. It can be felt under the integuments, in front, near to the coracoid process of the scapula. The man enjoys perfectly good health, he is entirely free from all pain, and he is enabled to do sufficient work to obtain a livelihood.

Remarks.—I have not brought this case before the Society without duly considering whether it might not be regarded of too trifling a nature, and scarcely worthy to occupy the attention of its members. But, although "decapitatio humeri" is not very uncommonly performed, and there are records of many such operations, we have only a few cases in which the results of those operations are subsequently detailed at a distant period of time. We are, in truth, greatly in want of such recorded experience, and to this end, and in the hope of inducing others to follow the example, I offer this case for comment and observation.

The age of the patient was somewhat advanced, and this circumstance might be regarded as a cause likely to militate against the success of the operation. The man, however, had always lived in the country, had enjoyed very good health, and his habits were very steady and quite free from intemperance in any way. The cause of the disease was an injury, which would lead to the inference that a particular spot in one of the bones forming the shoulder-joint was diseased, and that the mischief in them was not extensive. From local indications, it was quite clear that diseased bone existed, and that until the cause of the local disease was removed, there could be no hope of improving or restoring the constitutional nutrition of the patient. Indeed, this case seems typical of the class of cases in which the excision of the articular ends of bones should be undertaken; for, I believe, that in all cases and in whichsoever joint, when diseased bone exists, there is very little hope of saving the limb unless that diseased bone can be speedily removed. In the majority of cases in

which diseased bone does *not* exist, the cases eventually do well without an operation, and the limb is saved; it may be with a somewhat misshapen member. In the case related, a small piece of necrosed bone in the head of the humerus proved to be the source of the local irritation, which being removed, all pain and suffering immediately ceased, and the general health of the patient directly improved. Such good results as these would quite justify a repetition of the operation on patients of an advanced age, for this man was undoubtedly sinking from constitutional disturbance alone. But in progress of time a still greater advantage was obtained, for the man was quite able to follow his employment, and gain sufficient for his subsistence. The amount of elevation of the arm was certainly limited; but I am not aware that, in any case of this kind, the patient has regained the power of raising the arm above a right angle with the trunk, or above the level of the acromion scapulæ.

The following case occurred in Guy's Hospital in 1848, and affords an admirable illustration of the advantages to be gained by "decapitatio humeri:"

Excision of the Head of the Humerus, by the late Mr. Aston Key; *from Notes taken whilst the patient was in Guy's Hospital, by* Mr. Thomas Bryant, *Assistant-Surgeon, Guy's Hospital.*

Thomas C—, an engineer, was admitted into Guy's Hospital, December 20th, 1848. He was between thirty-three and thirty-four years old.

He had always enjoyed good health until four months since, when, whilst engaged at work in Prussia, he was pulling with great force, and strained his right shoulder. Eight days afterwards he observed a swelling in the upper part of the arm, and on the inner side. This increased to ten weeks since, when it was of the size of an egg. He could move the shoulder-joint, but was unable to work. When he returned to England, he experienced so much

pain in the shoulder that he applied to Mr. Cock, who lanced the swelling, and matter and some bodies like hydatids escaped. This afforded relief for about a week, and then acute pain was felt in the joint, which was very severe at night. Cupping over the joint diminished the pain, but after a few days had elapsed it returned, and he was admitted into Guy's Hospital.

There was considerable swelling over the right shoulder, and pain upon pressure over the same part. The joint could be moved freely, but it produced intense pain. There was a small fistulous opening in the skin, over the anterior edge of the deltoid muscle and tendon of the biceps, from which a curdy strumous secretion escaped. A probe could be passed along the sinus from this opening for about six inches towards the coracoid process.

January 30th, 1849.—Mr. Key made an incision from the coracoid process to the insertion of the deltoid muscle, and another along the posterior edge of the muscle. This flap was reflected upwards, the joint exposed, and the head of the humerus sawed off below the tuberosities. The articular surface was deprived of cartilage, and the cancellous tissue carious. The glenoid cavity of the scapula seemed to be healthy.

Profuse hæmorrhage attended the operation, and many vessels were ligatured. Sutures were used to adjust the edges of the flap to the surrounding parts, and the wound healed without an untoward circumstance.

The man left the hospital July, 1849, the wound nearly healed.

A drawing was made in February, 1856, seven years after the operation, and is in the museum at Guy's Hospital. Preparations of the head of the humerus removed in these two cases are preserved in the same museum. During the last six years the patient has been actively engaged at Messrs. Maudsley's, the engineers, and he has enjoyed excellent health.

ON

DISARTICULATION OF THE SCAPULA

FROM THE SHOULDER-JOINT,

WITH

REMOVAL OF THE ACROMIAL END OF THE CLAVICLE.

BY

GEORGE MATTHEW JONES, M.R.C.S. ENG. AND EDIN.,

SURGEON TO THE JERSEY HOSPITAL.

COMMUNICATED BY

F. C. SKEY, F.R.S.

Received Dec. 10th.—Read Dec. 14th, 1858.

THE operation which I am about to narrate to the Society, is one which is specially interesting from the fact of its having (so far as I can learn) been but twice performed; first by Mr. Syme, in 1856, and subsequently, as that gentleman informs me, by a practitioner in Ayrshire; but whether this latter operation took place before or after my own I have not ascertained.

In both these cases the patients died within three months of the operation, but not, I am informed, from anything connected with it, death being occasioned by some fortuitous circumstances. The opportunity of proving how far the motions of the arm could be restored was thus unfortunately lost.

Mary Ann C—, æt. 14½ years, had always enjoyed good

health, until attacked by the ailment which occasioned the operation about to be detailed. Her father and mother are living, and in good health; of their children one boy and two girls are alive, and three have died or have been born dead. The survivors are reported to be strong and healthy.

Twelve months ago she felt pain at the top of the left shoulder, which she supposed to arise from carrying a heavy child. Three days after the first attack of pain an abscess formed, which burst spontaneously. The inflammation was most intense over the upper part of the humerus; there was some fulness about the insertion of the deltoid muscle, and some matter escaped from an opening which was nearly on a level with the posterior fold of the axilla.

The constitutional disturbance was severe; but after a long course of tonic treatment and generous diet the general health improved. The opening, however, did not close, and it soon became evident that disease existed in the scapula; there being various small collections of matter which pointed in different directions, and sinuses through which portions of necrosed bone were discharged. It was at this period that I was called in.

I received the foregoing account from her previous medical attendant, and perceiving at once that the case was likely to be one of moment, I advised her becoming an inmate of the hospital, into which establishment she was admitted.

Every endeavour was used to increase the patient's strength, but without success; her health failed rapidly, and I felt that operative interference was imperatively called for. At this time there existed five large fistulous openings over the left shoulder: two communicating with the clavicle, one with the head of the humerus, one with the glenoid cavity, and one with the dorsum of the scapula, and bare bone was easily reached by the probe in each place. Several small fistulæ existed in the scapular region, which did not apparently lead to necrosed bone, but yielded an offensive discharge. The suffering in the shoulder and side was very severe; the textures covering the former were generally thickened, puffy, and tender to the touch. The countenance

was anxious and attenuated; the pulse 100, small and weak; the tongue furred; the bowels generally costive.

On the 10th of May, the girl being placed under the influence of chloroform, I performed the following operation. I first made an incision along the whole extent of the spine of the scapula, carrying it an inch beyond this in the back, then another incision to meet this along the posterior border of the bone down to its angle. The integuments were raised by careful dissection, and the whole bone was fairly exposed. Its periosteal investment was everywhere found thickened and pulpy, and so softened as to yield easily to pressure of the finger.

When removing the bone I carefully detached the subscapular muscle and other structures, and after some difficult dissection I succeeded in isolating the glenoid and clavicular ends of the bone, and removing it entire. The acromial end of the clavicle was found, for an inch of its extent, so softened and altered by disease as to render its removal advisable. Only one blood-vessel, the posterior scapular, required a ligature, several smaller branches being controlled by torsion. The operation occupied three quarters of an hour. Several sutures and straps of adhesive plaister were used to bring the edges of the wound into apposition; the deeper cavities, including those whence the glenoid fossa and angles of the bone had been removed, were plugged with lint. Before proceeding further it will perhaps be better to give a description of the appearance presented by the diseased bone.

The scapula was extensively diseased, and its characters almost destroyed. The glenoid surface and neck were entirely removed, and, with the exception of a small portion externally, no vestige of the spine remained. New osseous matter appeared in the position which the latter had occupied, but in this also necrosis was in progress; a deep fissure and chasm, indicating its former situation, extended across three fourths of the body of the bone. The inferior angle, in which the process of ossification was yet incomplete, was enlarged and hollow, converted, here and there into a tunnel

by overarching growths of new bone, and a large and deep furrow extended from this angle along the whole length of the anterior costa as far as the superior border: it was at one part continuous with the chasm, which has been already mentioned as running across the body, and occupying the site of the spine. The body of the bone presented, further, two deep perforations. From these various cavities sequestra of dead bone were removed: other portions in various stages of exfoliation were found contained therein. The anterior border of the bone presented a considerable amount of new osseous matter. The acromial third of the clavicle was found so soft and disintegrated as to require removal. The head of the humerus was healthy, and covered with its natural cartilage.

On recovering consciousness after the operation, the patient was found to be in a state of complete prostration; she retched violently and vomited, and had constant jactitation, restlessness, cold clammy sweats, and an almost imperceptible pulse. Mustard poultices were applied to the stomach, hot-water bottles to the extremities; stimuli of different kinds were freely administered, iced champagne, &c., but everything was rejected; the vomited matters were even larger in quantity than the fluid imbibed, while thirst was excessive, and the tongue parched and arid. The following day it became necessary to administer frequent enemas of strong extract of beef, with port wine; this system of support was persevered in for three days with apparently very trifling amelioration of the symptoms. At this time I gave two doses of chloroform, of twenty drops each, which procured a trifling relief; but the remedy which at last proved effectual in entirely checking the vomiting, and producing an almost voracious appetite, was the celebrated Jersey soup, made from the conger eel, which is at once nourishing and gelatinous. I have reason to believe that the sickness arose chiefly, if not entirely, from the chloroform; for after her admission into the hospital, she was so extremely nervous and sensitive at any examination of the shoulder, that in order to use the

probe it was necessary, on three different occasions, to put her partially under its influence. This was done each time at a different hour in the day, and equally at all hours was followed by vomiting. If this effect was produced by so small a quantity, we may naturally suppose the much larger one required for the operation calculated to bring about the same effect in an exaggerated degree. It may, therefore, be fairly inferred that to this, and not to the shock of the operation, may be attributed the affection which had nearly brought my patient to the grave.

May 24th, 9 a.m.—Has had several hours of uninterrupted sleep. Pulse still rapid and small; tongue less furred; thirst somewhat abated. The whole of the dressings, being completely saturated, were carefully removed, when it was found that, in consequence of the strain from the constant vomiting, the sutures had all given way, and no adhesion was apparent in any part of the wound. The head of the humerus was entirely exposed; jagged and abraded edges everywhere presented themselves. Ordered, tepid water dressings, to be frequently removed in consequence of the immense discharge. Nutritious diet, with quinine and wine.

25th, a.m.—Has taken much nourishment during the last twenty-four hours. Pulse much the same as yesterday; no restlessness, sickness, or retching. Tongue much moister; countenance by no means expressive of anxiety; the bowels have not acted since the operation; wound very offensive. An aperient enema was ordered to be given on the following morning if necessary, and to the shoulder very weak solution of chloride of sodium to be applied on pledgets of lint.

26th, a.m.—At 8 o'clock a large evacuation was produced by the enema. The night had been tranquil, but without much sleep. Pulse 88; considerable deposit in the urine; tongue improved. Discharge from the wound equally offensive and profuse as yesterday. Having on several occasions witnessed the benefit of an application of the following ointment, I ordered it to be applied on small

pieces of lint over the entire wound, and the whole covered with charpie.

℞ Cerat. Resinæ, ʒij
Bals. Copaibæ, ʒv
Sp. Terebinth., ʒiij. Ft. ungt.

27th, a.m.—The night has been good. A natural evacuation took place before breakfast. The patient expressed herself as comfortable and willing to eat anything. The urine was clearer; there was a great amelioration in the wound; the discharge appeared much more healthy and less fetid, in quantity much the same. Ordered, long adhesive straps, commencing at the collar-bone, to be passed round the arm and shoulder in order to afford all possible support.

June 6th.—From the last report to the present time a rapid and most gratifying improvement has taken place. Healthy granulations are appearing in all directions; the large vacuum is closing, and the head of the humerus is becoming covered.

It would be equally tedious and unnecessary to give an account of my daily morning visits, which, for the first week, were repeated every four or five hours during each day and night. Suffice it to say, in three weeks and as many days, she was able to leave her bed and walk round the garden, and at the end of another week from the operation she could sew without pain or difficulty; the discharge continued for about six weeks, but rapidly diminished; her appetite was always good, and her craving for food could scarcely be satisfied.

I have not stated the particular food I recommended this patient; suffice it to say that everything that was nutritious was unsparingly given her. Eight glasses of wine, and sometimes more, were allowed every twenty-four hours, in addition to three pints of porter.

At present (November) she can raise her arm twelve inches from her side, and can, with very slight assistance, support it horizontally from the body. She can raise the

hand to the opposite shoulder or to the mouth with ease, but not to the top of the head; she can put her arms behind her, can lift a large and heavy hospital register, and can scrub the floor, or make her bed.

There is a decided falling of the shoulder, but by no means such as amounts to deformity; there is no wasting of muscular substance on the chest or back, and when she is dressed, it would scarcely be apparent that any serious operation had been performed on her. The deltoid is of full size. The cicatrices of two incisions remain, each of about six inches in length. A small old cicatrix also remains over the scapular end of the clavicle. The head of the humerus is easily felt moving freely in its new bed, and not the slightest pain is experienced on any amount of motion. Up to this time the range of this motion has steadily increased, and with the exception of the movements for which the scapular origin of the deltoid is indispensable, it may be confidently expected to increase still further, and nearly to equal that of the other arm.

The conclusion at which I have arrived from the account of Mr. Syme's case, as well as from my own experience in the present instance, is that the danger from hæmorrhage is much less in removing the whole of the scapula than, as in the case recorded by Mr. Liston, in the removal of only a portion; and in future no doubt would exist in my mind as to which would be the preferable operation, should the necessity for either come again under my notice. It may be remarked that in Professor Syme's case the progress towards recovery commenced much earlier than in my own; his patient, however, had the advantage over mine in the previous state of the limb. I had to contend with numerous sinuses, nasty sores, and decayed portions of bone, so that when the incisions were made they presented jagged instead of smooth edges, the whole being in fact a disorganized mass, while the surfaces in Mr. Syme's case were comparatively healthy.

DESCRIPTION OF PLATE I.

The bone is seen on its dorsal aspect, but slightly turned, in order to give prominence to the inferior costa.

The structure of the whole bone, particularly in its front, is swollen and spongy, the consequence of increased activity of growth for replacing the necrosed portions by new bone.

Along the inferior costa, and reaching beyond the upper part of the scapula, an elongated, hollow case or shell of bone may be observed. Its extent and direction are indicated by a piece of whalebone, one end of which is marked A, and the other A*, passed along it. The shell formerly encased original bone, in the state of necrosis, and has been formed by the separation and expansion through the process of new growth of the two laminæ composing the scapula. Near the former site of the glenoid cavity and cervix, a part of the interior of the case has been exposed to view, owing to the walls having been partially absorbed, and a cloaca thereby formed, at that part.

B.B'. Whalebone passed through a cloaca, which conducts into a short branch, or irregularly expanded portion of the above-described cavity.

C. A wide gap, extending about two thirds across the body of the scapula, in the part formerly occupied by the spine; which, except a small portion of its root in front, has been destroyed by necrosis.

The fragments arranged round the principal figure, fourteen in number, are the various sequestra.

B
A'
C
B'
A

CASE

OF

ENCEPHALOID CANCER

AFFECTING A TESTICLE WHICH HAD BEEN RETAINED WITHIN THE CAVITY OF THE ABDOMEN.

BY

GEORGE JOHNSON, M.D. LOND.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; PHYSICIAN TO KING'S COLLEGE HOSPITAL.

Received Nov. 2d, 1858.—Read Jan. 11th, 1859.

THE subject of the following history was C. D—, æt. 27, a well-developed, muscular man, of active habits, a zealous boater, cricketer, and sportsman. When a child he was delicate, but after the age of eleven he enjoyed robust health, until he overworked himself in preparing for a scholarship examination at Cambridge, when he became dyspeptic and nervous. He first consulted me for these symptoms in the year 1853. He gradually recovered, worked for his degree, and graduated in honours. He then continued to reside at the University as a private tutor. The second occasion on which I heard of him as a patient was in the month of April in the present year, 1858, when he wrote to me from Devonshire. His letter is dated April 9th, 1858; and the following is his own account of the commencement and progress of the illness for which he then consulted me:

"Last September, while out shooting on a very fatiguing beat on a hot day, an uneasiness in my *right* side,[1] which I had felt for a short time before, grew into such intolerable pain, that I had to knock up for a couple of hours. It went off, and I finished the day's sport without suffering much more pain. For a day or two afterwards the pain was severe. A country medical man assured me it was muscular, and applied hot fomentations. The sensation was that of an aching of the *bone*, and did not appear to be near the surface. Up to the middle of January this uneasiness continued incessantly; sometimes, but not often, becoming positive pain. I rowed during the whole of November, but this did not cause any increase of uneasiness, on the contrary, I think it was less than at any other time.

"In January I went to two balls, and after the last the muscle above the hip was much swollen, and the pain increased so much, that I consulted a surgeon in Cambridge. He, too, called it muscular; but by the middle of February the pain became so bad, that for four or five hours at a time I could only find ease by sitting in a crouching posture. This would be the case two or three times a week, and I do not exaggerate in calling the pain at such times absolute agony. I also had frequently a severe contraction of the rectum, often expelling its contents involuntarily. The medical man changed his mind, and thought the *caput coli* was at fault, and gave me mild aloes pills.

"The urine is slightly high in its sp. gr., but clear and of good colour. The pain of passing water is very great at times. *Once*, while the pain was very intense, I passed some urine which left a thick deposit of mucus, and was quite turbid while warm.

"Just before I left Cambridge the surgeon told me that he had ultimately made up his mind that the pain was attributable to the passing of renal calculi along the ureters. But I have never had any pain in the kidneys."

[1] The seat of pain was just above Poupart's ligament on the right side.—G. J.

My first impression, on reading this account of the patient's symptoms, was that he had renal calculus; and I thought this the more probable from my knowledge of the fact, that, when he was under my care five years before, his urine contained oxalate of lime in great abundance. I now wrote and asked some questions which were suggested by this view of the case. What these questions were will be evident from his reply, which is dated April 11th, 1858, and which I give in his own words:

"As you have asked me questions regarding the retraction of the testicle, I will at once make to you the confession, distressing to me to make, that I have no *right* testicle, nor do I remember ever possessing one. The left is of the ordinary size, and so far as I can judge the proper and healthy operation of the sexual organs is not impaired by this defect, at least I have never experienced any inconvenience on this score. It is painful to me to impart this secret, which I have hitherto guarded with the greatest care, nor is there a soul besides yourself who is at all aware of it. True it is, that we have all of us 'a skeleton in the cupboard' of some nature or another. There has been no retraction of the testicle of any consequence, nor has there been pain in the right thigh. My general health is pretty good; but having led a very sedentary life during the past winter, I am not in a *rude* state of health. Muscles rather relaxed and flabby. The pain has never produced a feeling of nausea. To-day I am upright again, but there is an aching towards the extremity of the glans penis, which I have not felt till within the last few days. I have now, I believe, answered all your questions."

On the 18th of April he again wrote as follows: "I inclose some sediment that I passed last night. The pain was worse yesterday than I have felt it for some time. I drank three hot glasses of weak gin and water last evening, and passed only about three fourths of a pint, after an interval of at least ten hours since the last time of passing any urine. It has given me pain for some days past, as I told you, to pass any urine at all. To-day, with the

exception of a slight uneasy feeling at the point of the hip, I feel as well as I ever did in my life. Yesterday not able to walk one hundred yards, and doubled up. To-day able to walk a couple of miles, which I am just going to put into practice, and perfectly upright. Surely I am not wrong in attributing this change to the passing of the sediment."

The sediment here referred to was composed of the ordinary urate of ammonia, with numerous small octohedra of oxalate of lime. There was no trace of albumen in the urine, nor did the microscope reveal the presence of pus or mucus.

Up to this time I had not seen the patient, but on the 17th of April he came to me on his way from Devonshire to Cambridge. His general appearance did not indicate any serious organic disease; there was no emaciation, and his limbs were firm and muscular. The pain of which he complained appeared to be near the bladder on the right side, but there was no irritability of the bladder, and there seemed no reason to suspect the existence of a calculus either in the kidney, ureter, or bladder.

The recti muscles appeared to be unusually firm and rigid; but with this exception I detected nothing abnormal in the state of the abdomen after a most careful examination, and I could form no opinion as to the nature of the disease.

May 31st.—I saw him again in London. In the mean time he had been suffering more or less constantly from the pain low down in the right side of the abdomen. On the 21st of May he had joined in a cricket-match; and while engaged in the game, he was suddenly seized with unusually severe pain, which compelled him to lie down for some time. He was then carried home and placed in a warm bath. From that time the pain has been more severe, and his condition has very much changed for the worse. He now walks in a bent position with much pain and difficulty; he has lost flesh considerably, has a pale, anxious, haggard look, and is convinced that his disease will, as he says, "end in the churchyard."

He returned to Cambridge, where he continued to lose flesh and strength, and to suffer from severe and almost constant pain. The nature of the disease being still doubtful, I gladly acceded to the patient's wish to have another opinion upon his case; and I arranged a consultation with Dr. Bright on the 12th of June, the patient again coming up from Cambridge for the purpose.

In giving the history of the case to Dr. Bright, I told him that I had ventured to make a guess at a diagnosis. Connecting all the facts which have already been mentioned with one not before alluded to, namely, that the patient's mother had died of cancer of the stomach, it seemed to me not improbable that the right testicle, retained in the abdomen, had become the seat of malignant disease, and that in this might be found the explanation of the long-continued pain, and of the recent rapid emaciation. We then proceeded to examine the patient's abdomen, and Dr. Bright pointed out, what was now sufficiently evident, that just above Poupart's ligament on the right side, there was a growth or deposit either in the abdominal wall or immediately behind it—this portion of the abdomen being hard and resisting under pressure, and yielding a dull sound on percussion. There was besides, considerable fulness of the whole lower part of the abdomen. Dr. Bright's opinion was, that there was a deposit in the abdomen, perhaps cancerous, and he thought it possible that the retained testicle might be, as I had suggested, the primary seat of the disease. From this time the progress of the case was very rapid. After the consultation with Dr. Bright the patient returned to Cambridge, where he remained about a week; he then went to stay at the house of a relative about ten miles from London, where I was asked to see him on the 29th of June.

I found him wonderfully altered, very pale, and much emaciated. He was in bed, though he had been downstairs the day before my visit. The tumour in the abdomen had greatly increased, extending now beyond the median line, and considerably above the umbilicus. It was quite im-

moveable under pressure, and appeared to be closely adherent to the abdominal wall. The whole surface of the tumour was dull on percussion, but there was resonance in the region of the ascending colon. The greatly distended abdomen had very much the appearance observed in a case of large ovarian cyst. During the previous day or two there had been occasional bilious vomiting, which he had encouraged by copious draughts of warm liquids.

I did not see him again alive. He became rapidly weaker, and died on the 7th of July, retaining his consciousness to the very last.

Forty-seven hours after death I examined the body with my friend Mr. Spurrell, who had been in daily attendance upon the patient after he left Cambridge.

The body was much emaciated. The tumour had increased considerably since my visit on the 29th of June; it now extended far over to the left side, and as high as the epigastrium. It felt firm and hard through the wall of the belly. The whole anterior surface of the tumour adhered to the wall of the abdomen, and there were some adhesions about the right iliac fossa. The adhesions were broken down without much difficulty, and the tumour was removed. When *in situ* it occupied a large part of the abdominal cavity. The small intestines were pushed upwards and to the left, and the liver was pushed up beneath the ribs; it formed no adhesions to any of the viscera. In removing the tumour about four pints of dirty grumous liquid escaped from some of the large cysts contained in its substance. The weight of the mass after the escape of this liquid was sixteen pounds; its original weight could not have been much under twenty pounds. Its dimensions were as follows: length, fourteen inches; breadth, twelve inches; thickness, from three to six inches. At the under surface of the tumour was a projecting body, about the size of a duck's egg; its surface was smooth, covered by a bluish-white capsule, which above was spread out and gradually lost over the tumour. The vas deferens, quite normal, passed to the back of this body, and some

fibrous tissue went from its anterior surface to the internal abdominal ring. This body was evidently the right testicle, which above was spread out into the large tumour already described. On section, the testicle was found degenerated into a cancerous mass. My friend Dr. Andrew Clark examined it, and found in it distinct traces of its original tubular structure. The testicle and the whole of the diseased mass into which it had grown had the usual characters, both to the naked eye and under the microscope, of fungus hæmatodes. The tumour was, for the most part, a soft, solid mass, but it contained many cysts, varying in size from that of a hazel-nut to that of an orange. One cyst was as large as a good-sized cocoa-nut. Many parts of the diseased mass were infiltrated with blood.[1] All the other viscera in the abdomen were quite healthy. The chest was not examined.

There is yet one feature of this remarkable case which is worthy of notice—namely, the extreme pertinacity with which the patient kept the knowledge of the non-descent of the testicle from every one but myself. Mr. Baxter, of Cambridge, to whom I am indebted for some particulars of the case, informs me that, although Mr. D— was aware of the doubt which he and his partner, Mr. Lestourgeon, entertained respecting his disease, his constant reply to any question relating to the state of the testicles was that "he had never had anything the matter with them;" and Mr. Spurrell, of Bexley, who attended the patient during the last three weeks of his life, and to whom I had communicated what I knew of his history, could never, by indirect questioning, elicit from him any acknowledgment of the peculiarity which had caused him so much misery.

Many cases are on record in which a testicle retained in the groin or in the inguinal canal has become the seat of cancerous disease. Several instances of this are related by Mr. Curling, in his valuable treatise on the Diseases

[1] I have a microscopic specimen prepared by Dr. Clark in which portions of the tubuli testis are still visible.

of the Testes. One such case is recorded, and others are referred to by Mr. Arnott, in a communication published in the thirtieth volume of the 'Transactions' of this Society.

Hitherto I have neither heard nor read of any case similar to the one whose history I have here narrated. The chief features of the case were the enormous size attained by a cancerous testicle which had been retained within the abdominal cavity, the rapid progress of the disease, especially during its later period, and the severe pain which attended it.

CASES

OF

RE-FRACTURE OF BONE;

WITH OBSERVATIONS.

BY

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Received Nov. 10th, 1858.—Read Jan. 11th, 1859.

I VENTURE to bring before the Society the report of a few cases of more or less interest to the surgeon, viz., of re-fracture of bone, to which I have added some explanatory observations.

The subject of re-fracture of bone as a curative process in surgery has, perhaps, not sufficiently occupied the attention of the profession. Although it is true that, by virtue of a higher-classed surgery, the necessary resort to re-fracture must be comparatively rare, we have yet no exemption from liabilities to failure, the result of eccentric varieties of fracture, and constitutional peculiarities both of a moral and physical character. The difficulties incidental to the management of such cases can hardly be adequately tested by the statistics of our hospital practice, whether metropolitan or provincial; because such institutions, from the abundance of their resources, may be supposed to possess all required appliances essential to treatment under

every circumstance of difficulty and complication. Notwithstanding the ample supply possessed by our hospitals and other large institutions, the varieties and complication of splints, and other agents employed for the purpose of maintaining exact apposition between the broken ends of a bone, it will not be denied by men of even moderate experience, that cases of extreme difficulty are not of very infrequent occurrence, which tax to the utmost the ingenuity and the perseverance of the surgeon. We occasionally meet with combinations, in the same case, of oblique fracture of the shaft of a long bone, large muscular power, and more than ordinary irritability of the nervous system—three conditions which collectively set at defiance every attempt to maintain the entire apposition of the broken ends of the bone, coupled with that perfect immobility of the limb, on which alone complete success must depend. Another element of difficulty is occasionally found in extreme irritability of the integuments. In such cases, the subject of fracture is unable to bear with impunity the requisite pressure caused by the splints, and ulceration follows with remarkable rapidity on one or more projecting or salient points of the limb, compelling a change of apparatus, generally from a perfect to a less efficient form of mechanism.

Of this difficulty I had a remarkable example in St. Bartholomew's Hospital, during last summer, in a man, æt. 40, who sustained a fracture of the middle of the thigh-bone. The bones being adjusted, a long splint was applied to the limb by means of the ordinary bandages; but, in the course of a few days, he complained of such severe pain, caused by the pressure both of the splint and bandage, that I was compelled to order their entire removal. The parts subject to pressure were found to be congested with blood, and extensive ulceration followed in the region of the hip, groin, and foot; all restraint was rendered nugatory, and the man recovered with an overlapping bone and a short limb. Nor is this the only case of this peculiarity of constitution that has come under my observation. I may

allude, among other examples of the evil results of intolerance of pressure on the skin, to a case of popliteal aneurism, which I essayed to cure by means of the pressure of a tourniquet. Before commencing the *permanent* resort to the instrument, I applied the tourniquet over the femoral artery, in what is termed Scarpa's triangle, with a slight tension only of the screw; but, within forty-eight hours, the whole surface became erythematous, the limb swelled, and I was compelled to tie the artery. Moral wilfulness also is no very infrequent obstacle to complete recovery from a fractured bone. In the summer of 1857, I had a man with this attribute under my charge in St. Bartholomew's Hospital. He had broken his thigh-bone about the middle. He also was treated with a long splint. Finding the perineal bandage disagreeably tight, he relaxed it during the night. The house-surgeon remonstrated with him, but in vain. On the following day he untied the bandage, and set the splint loose. In vain I assured him that his recovery depended absolutely on his tractability, and that without implicit obedience he would become a cripple for life, but he persevered in the management of his own case, the bone united with the limb three inches short of its original length, and he left the hospital.

In some examples where the fracture takes place by a long obliquity, and the fractured surface is more than usually smooth, there remains nothing but the force of the extending splint, combined with its lateral pressure, to maintain the broken surfaces in apposition. But an extending splint, unless applied with unusual tension, contends imperfectly against the persistent influences of muscular contraction. The consequence is that the contraction of the bones, especially in persons in whom the ossific reparation is more than usually slow, continues to increase, while the altered relation of the bones is so insidious as to escape observation, and the final union may exhibit the bone abridged to the extent of one or two inches of its normal length. However perfect may be the measurement of the limb at the date of the first application

of the splints, the retraction to the above extent is complete, and the union is firm.

I do not mention these cases of imperfect recovery from fracture with a view to the appropriateness for re-fracture, but rather with that to their rejection. I would select such examples of failure as are attributable to causes, incidental to the first fracture, but avoidable on the second; and inasmuch as both constitutional irritability and moral unfitness for personal restraint would be rather aggravated than mitigated by protracted physical suffering, it must be obvious that such are not cases to sustain a repetition of similar and possibly even more critical restraint than appeared to be required by the former injury. The case may be one of inherent difficulty founded on the combination I have alluded to, in the form of great obliquity of fracture, occurring between the ages of twenty-five and forty-five, a large muscular development with a natural intolerance of restraint; the extension may be inadequately made; the mechanism, though sufficient to the end of maintaining complete apposition of the broken surfaces for ordinary occasions, is insufficient for this. The measurement of the limb made by coarse tape, or by any material at hand, however unsuitable, to determine the exact adjustment of the bone, finds a defect of apposition of half an inch or more, which, it may be argued, if not increased by further retraction during the treatment, may be expected to restore a good, serviceable limb without apparent lameness in the future gait of the patient. If to these conditions be added inexperience in the application of splints, coupled with imperfect mechanical knowledge, we have the elements of a permanent retraction of the bone to any amount, whether in the case of fracture of the bones of the lower extremity so slight as not to affect the gait in walking in any appreciable degree, or sufficiently great to cause permanent and injurious lameness for life. In the instance of the upper extremity, I have been led to resort to re-fracture of the bones of the forearm in cases, in which retraction of both bones (itself an evil, if taken alone,

of no great moment) has been accompanied with loss of muscular power, whether of flexion or extension, of inability to rotate the arm, or of permanent pain resulting either from pressure on or displacement of one or other of the nerves of the forearm, or, possibly, from some entanglement of muscular or fibrous tissues in the uniting medium of the fractured bones. Surely on such conditions it is warrantable to retrace our steps towards the condition of the patient at the date of the first fracture, provided that object be attained without *difficulty*, and its almost necessary concomitant, *danger*.

That actual re-fracture may be attained without the presence of either of these objectionable elements I think I shall be able to show. It does appear that the uniting material of a bone, at the expiration of three, four, or even five months, is capable of being separated by lateral pressure with a comparatively small effort. The results of the experiments on animals instituted by the great French surgeon, Baron Dupuytren, led him to the inference that a greater quantity of hard matter, whether bone or not, was deposited about the broken ends of the bone than was required for ultimate use; and the examination of the evidence obtainable from preparations of the uniting bones of animals, undoubtedly exhibits, on all occasions, solid masses of firm substance around them. It has yet probably to be determined whether the process first described by Dupuytren may not apply as the rule in those cases, in which the attempt at motion between the bones prevails, or whenever we fail to effect that entire and absolute rest of a broken limb, which the moral restraint as well as the higher sensibility of man will commonly, if not invariably, command. It is almost impossible to ensure perfect immobility of the broken limb of a dog even from the hour of the fracture, and the consequence is that under the best management the deposit of bone around the fractured ends is large in quantity, as if coarsely thrown down in the neighbourhood of the fracture rather than in a direct line between the fragments. Very commonly the bones, not-

withstanding the superfluous amount of bony deposit, fail to unite, and a permanent false joint is the result.

The practice by Dupuytren of attempting the readjusting of fractured bones imperfectly united at a considerable term after union was completed, is no doubt in the recollection of the surgical members of the Society. Many cases are cited as examples of resetting bone by means of gradual and permanent extension and lateral pressure, while direct rupture of the uniting medium is strongly deprecated. No one will doubt the greater appropriateness of simple and gentle means employed in disuniting a bone surrounded by soft parts, if the simple means suggested will effect the disunion; but if gentle efforts will not attain the end, the question arises, are we justified in resorting to more powerful agency in order to obtain separation of the bones? I believe we are. The means resorted to by Baron Dupuytren were gradual extension and lateral pressure. If we examine a preparation of a broken femur at the expiration from the fracture of seven or eight weeks, in which overlapping of the bones has occurred to such an extent as to require, or even to justify, an attempt at separation, supposing the limb be shortened to the extent of an inch and a half or two inches, it is obvious that lateral pressure must be perfectly inefficacious, and that any attempt to lengthen the limb by permanent extension must be nearly equally so, because, as the bone has at that time acquired sufficient strength to support the weight of the body without separation of the uniting medium, we may reasonably infer that no less powerful extension than the equivalent to that weight could effect any change in the new structure, and probably a great deal more than this force would be required to elongate it. M. Dupuytren assigns a period of sixty days, at the expiration of which it would be imprudent, he considers, to attempt the separation. At the same time he publishes cases of successful treatment applied to fractures considerably exceeding the above term. These examples, however, refer either to cases of imperfect union, or to such deviations from perfect recovery as are

characterised by deformity caused by lateral distortion, rather than by deficiency in the normal length of the affected limb. These are not cases of overlapping, but merely of union of the broken extremities in an indirect line; the points of contact, being limited to the extremities only, are capable of separation by very slight force.

But I am the advocate of a proceeding of a larger kind. I believe bones which have united to any length by lateral apposition of the entire diameter of the shaft, and in which the spaces formed by the contact of the two cylinders are filled with fibro-osseous matter possessing firmness sufficient to support the superincumbent weight of the body, are susceptible of disintegration by a judiciously applied force to the advantage of their replacement to their natural relations prior to fracture, and without injury to the soft structures around them.

I place the warrant for re-fracture of bones imperfectly united, with overlapping to any moderate extent, on the grounds of its practicability, of its safety as an operative proceeding, and of its indispensability to the perfect utility of the limb involved.

First, with respect to its practicability. We are not to gauge the density of the uniting medium of a fractured bone, or to infer its absolute osseous nature by its power of resistance to the superincumbent weight. If this material be examined at the expiration of even four or five months, when, under the slight protection of the pressure of a bandage, a man has resumed, in a limited degree, his daily occupations, it will often be found to be composed of fibrous tissue, in which the deposit of bone is yet limited. My observation leads me to believe that the perfect process of reunion is greatly protracted in examples of imperfect adaptation of surfaces, and that we are not justified in calculating on an exact rate of progress in cases presenting various and dissimilar conditions, including every variety of fracture of which bone is the subject. The difference of time may extend over a period of several weeks. Again, in examples of overlapping in the long cylindrical bones,

it would appear that the two portions, though laterally united, fail somewhat in nutrition, and become more or less atrophied; the walls become reduced in thickness, and the medullary cavity in diameter. The extremity of each portion is closed, and presents the appearance of a truncated cone. It may be perfectly true that the union of such a fracture is compatible with moderate exercise, and that the limb will support the weight of the body without pain or discomfort of any kind, beyond that arising from the defect of gait which is dependant on the inequality in the length of the two limbs. But it does not follow from this, that it will yield to lateral force. In the upright position the resistance is made by the whole length of the uniting medium. By bending the limb at the line of union we have the resistance only of the transverse diameter. If this union be susceptible of rupture, it is effected without great difficulty on the part of the surgeon. The practicability of re-fracture of a bone in any given case can only be brought to the test of experiment. It is impossible to lay down any inflexible rule of conduct. We may be confident of our ability to effect separation in some cases, and our inability in others. We cannot define the exact line on which success and failure meet. The power to re-fracture is obtainable on different conditions in different cases. It depends on age, possibly on sex, on the extent of surface involved, on the duration of the healing or uniting process, and finally on the tact as well as the force of hand employed in the operation; but that, on ordinary conditions, it may be effected for many weeks, or even months, after the restoration of the limb to careful exercise, there appears to me no good reason to doubt.

Secondly, with respect to the question of safety, two points are involved. 1st. The reasonable probability that the bone will break at the site of the original fracture, and at no other part. 2dly. That the separation may be effected at the site of the original fracture, without injury to the soft parts around. I know but of one bone in the human body coming within the required conditions, which

can be broken, even in its dried state, by the hand of man employed with all its muscular force, viz., the fibula. I have seen a powerful man entirely fail to break a single radius, on which his muscular force was immediately directed. Is it possible to apply a similar force to a bone surrounded with soft structures on every side? Long before such a force could reach the bone, the skin would give palpable evidence of violence by laceration. In a case in which, after the most vigorous efforts, I failed to re-fracture a femur which had united for nine months, the man sustained no injury beyond a bruising of the skin, from which he entirely recovered in two or three days. Moreover, the broken ends of the bone become rounded off, and there remain no sharp or prominent angles, or spicula, which our natural apprehension of danger might point to, as fruitful sources of injury to surrounding vessels or nerves, and the separation is, of course, made with due consideration to their possible proximity. The effort made, and the force applied, are gradual and deliberate, and it is better, should the bone yield by pressure upon it *in one direction*, to break down the connecting medium by slight extension *in all*, and that the broken bone be carried around the circumference of a small circle rather than forced in the single radius of a larger one. It should also be rotated on its axis in the case of a single bone, as the femur or humerus.

The experiments of M. Dupuytren in this path of surgical science obviously refer to cases of deformity arising from a defect in the direction of the broken bone, or in which an angle is formed at the point of union. He recommends the employment of "a graduated force," to be maintained, and gradually augmented. Now, as my experience proves the practicability of the re-fracture of bone many weeks after the imperfect union has proved sufficient to support the weight of the body, it is obvious that no application of an extending force of less amount than one hundredweight and a half could be available to any useful purpose. How is such a force to be applied? Could it be resorted to for even a brief period without suffering and danger? M.

Dupuytren quotes the authority of Dr. Jacquemin in favour of the extension of the limit of sixty days, beyond which he would deem the attempt unwarrantable. But I have endeavoured to show that it is impossible to fix a limit; that the nature of the uniting material is dependant on age, on sex, on health, on immobility, and on the mechanical relation of one fragment to the other; and I have broken a femur, firmly united, and without danger, at a date the double of that which M. Dupuytren has named as the utmost limit at which the attempt would be justifiable, and I have broken it by the very means which he has deprecated as dangerous.

Thirdly, with respect to the question of interference in these cases, it can only be determined by the requirements of any given example. If, the fracture having occurred in the upper extremity, the imperfect union of the bones have compromised the movements of the arm in pronation and supination, or materially impaired the actions of the fingers or hands, or if in the lower extremity a considerable degree of lameness is the result, whether consequent on obliquity of the bone or abridgement of the length of the limb,—in either case, be the interval from the accident long or short, be it one month, or four, five, or six, I can find no objection under ordinary conditions to the attempt at re-fracture with a view to readjustment. The limit as to time must, as I have said, be indefinite; the practicability to accomplish the separation of the united bones without injury to the soft structures of the limb constitutes the sole, but as I think the sufficient, guide to its warrant. Short of such an occurrence, any degree of force may be applied, in the full assurance that should the bone break, it will break at the point of fracture, and in no other part. I would suggest, if common discretion did not otherwise dictate it, that the force be applied with all requisite care, gentleness, and deliberation. Its direction would be determined rather by the relation of the bones to each other, than by that of the vessels of the limb. We have, in the attempt to reduce old dislocations, sufficient evidence that

the large vessels of a limb may be made liable to a good deal of violence with impunity, that they may be subjected to considerable displacement, to retraction, and again after the expiration of several months to elongation, without damage to their structure or function. In the case of old dislocations of the femur on the dorsum ilii, we elongate the femoral artery to the extent of its abnormal retraction. This would probably be equivalent to from one to two inches; and, in the case of the humerus in dislocation under the pectoral muscle, to that of one inch. But I know of no example of injury sustained by the main artery of either limb in the attempt at restoration of the dislocated bone to its normal relations. It will hardly then be asserted that the liability to injury of the arterial system of a limb from the required elongation presents any obstacle to the operation of re-fracture; while, if we reflect on the nature of the union of overlapped bones, and recollect how completely the rough projecting surfaces of bone are rounded off by time, we can scarcely admit the liability to injury to the vessels from their cautious separation.

The act of disuniting the bone is effected by slow laceration rather than by a snap or fracture. It is not a sudden but a gradual process, requiring persistent rather than sudden force. It is the act of tearing rather than breaking, the name of which implies the fracture of hard material, whereas I have endeavoured to show that the uniting medium, though sufficient for the purposes of locomotion, is yet soft and lacerable.

With respect to the requisite elongation, on all accounts it should be effected by a great power gradually applied, and that of the compound pulleys is desirable in cases in which the retracting force of the muscles is considerable. In children, or in the emaciated limb of an adult, pulleys are not essential. The extension, however, should suffice to restore the limb to its normal length, and every appliance required to maintain the bones in exact apposition should be within reach, and prepared for instant application.

CASES.

The first case to which I shall refer is recorded in a paper by Mr. Wiblin, of Southampton.[1] The bone involved was the right femur. The man's age, 23. Eleven weeks had elapsed since the fracture. The bone was firmly united, as may be inferred from the above interval. The limb was two inches short of its natural length. The man's gait was very imperfect. Having placed him at the end of his bed, laid the thigh over a firm piece of board, and made every requisite preparation for retaining the bones in the natural situation, chloroform was administered. The patient being rendered perfectly insensible, I gradually leant my weight on the knee, and the separation was audible to persons around. I then rotated the bone in all directions, until the separation was complete. Pulleys were applied by means of a strap fixed round the ankle-joint, and gradual extension made for three quarters of an hour, before the normal length of the limb was restored. His recovery was complete.

Case 2.—A man, æt. 40, was treated in the Casualty Ward of St. Bartholomew's Hospital for oblique fracture of both bones of the right forearm, about halfway down. He declined admission as an in-patient. In consequence of his negligent attendance at the hospital, the bones overlapped, and were fixed in partial supination. He lost the power of closing his hand, and he suffered severe and continuous pain at the seat of fracture. I gave him an opiate liniment to apply frequently. The bones had apparently united by the thirtieth day, but both the local pain and the distortion of the limb increased, and at the expiration of the eighth week I proposed to him the readjustment of the fracture under chloroform. He declined this offer, and I saw nothing more of him for several weeks. However, the pain continued, and he determined to submit to the opera-

[1] 'Lancet,' July 21st, 1855, p. 49.

tion. This I performed at the expiration of the seventeenth week, either on or about the 120th day. The patient having been put under the influence of chloroform, I drew the arm over the corner of the operating-table, and carefully applied a portion only of my weight. The bones separated without any difficulty. Extension was made slowly but efficiently, and the forearm fixed by lateral splints in slight pronation. The pain subsided immediately, the movements of the arm were fully restored, and the man's recovery was perfect.

Case 3.—I relate the following case, in which my attempt to break the bone failed, for the purpose of showing that great muscular power may be employed on the surface of a limb without injury to the soft structures beneath.

In this example, one of fractured thigh, the overlapping was very great, and the obliquity considerable. I have alluded to this man's case as that of imperfect union of bone from wilful interference and great intolerance of restraint during the treatment. He applied to me at St. Bartholomew's Hospital, after an absence of several months, intreating me to give him some relief. He consented to my proposition of the attempt to break the bone, which, considering that more than nine months had elapsed since the accident, did not promise a very favorable issue. Long as was the period since union had been completed, it did not appear probable that injury would arise from the attempt, and he was placed under the influence of chloroform. I employed the full force of pressure in every direction, by every warrantable means, but could make no impression on the fractured bone. He sustained, however, no injury through the attempt, and he left the hospital on the fourth day.

Case 4.—My attention was called to the case of a gentleman, æt. 46, who had broken his thigh, about the middle, by a fall. By some mismanagement in the placing of the bone at the time, the length of the limb, when union was completed, was found reduced to the extent of an inch and

three quarters. The lower fragment rode forwards, and its projection remained very prominent under the quadriceps muscle. I saw him on the twenty-fourth day from the date of his first supporting the weight of his body on his limb. To all sensation the union was perfectly firm, and he moved upon the limb without apprehension or pain; but the inequality in the length of the two limbs rendered his gait very unsightly to others and irksome to himself, which was further increased by the circumstance of the bone being distorted by the obliquity of the union. Another evil arose from the present condition of the limb, viz., inability to flex the thigh to its fullest extent on the pelvis, in consequence of the rectus muscle being implicated in the fracture.

On the seventy-fifth day from the fracture I separated the bones at the point of fracture. The operation, if it merit the name, performed of course under the influence of chloroform, was effected, as in the former thigh-case, by bringing the fractured part to the edge of a thickly padded board of wood placed on the end of the bed. I leant my weight on the front of the knee, pressing the lower fragment backwards. The force was not very great, at least was greatly within what I retained the power to exercise, had it been necessary. The separation was completed by the rotation of the leg in the entire circle. Pulleys were employed to elongate the limb to its normal length, which was not accomplished within a period of forty minutes. A long splint, a front and inner splint, were applied with all requisite severity of tension, and were retained in that condition for a fortnight, and finally removed at the expiration of the seventh week. The bone had now fully united at its broken surfaces, and the normal length of the limb was restored. The result fully justified the treatment adopted, and rewarded the patient for his long period of confinement and deprivation.

Case 5.—A young man, æt. 22, sustained a fracture of both bones of the leg. On the final removal of the splints

on the forty-sixth day, a gum and chalk bandage was applied, which was retained on the leg for a period of two weeks. Up to this date no measurement had been made, and no suspicion arose that the union was imperfect. I saw the patient in the eleventh week from the accident. The leg was shortened to the extent of an inch and a half, the upper end of the tibia projected forwards, and the lower end extended obliquely outwards so much as to render it difficult to throw the weight of his body on the flat sole of his foot. I had the patient placed under the influence of chloroform, broke the bones, extended the leg by the force of the pulleys, applied a long splint, and a second splint on the inner side of the leg. The union of the bones is firm; and at the expiration of two months from the date of the operation, the man walks firmly and well on the affected leg.

Case 6.—A child, æt. 6, sustained a fracture of both bones of the leg. The management of the case was undertaken by a female friend of the child's mother, who, having performed the functions of a nurse in a large hospital at a former period of her life, undertook the charge of the child. She applied splints, or the substitute for splints, on each side of the leg, and retained them in position for thirty-eight days. On the forty-fifth day I examined the leg, and found the tibia greatly bent backwards, forming an angle at the point of fracture, and the fibula overlapped to the extent of nearly an inch. The opposite limb was well formed, healthy, and thoroughly developed. When the child had been brought under the influence of chloroform, I broke the uniting medium, and extended the leg to its primary length. Two side splints appeared all that was required, and at the present date of three weeks from the re-fracture the child is progressing favorably towards its recovery.

Case 7.—A boy, æt. 15, was sent to me, whose right leg was fractured at about one third of its length from the ankle-joint. The fracture, which was caused by a cart-wheel passing over the leg on ploughed land, was very

oblique, and involved both of the bones. He was placed in bed, and lay, without any attempt being made to adjust the bones, for several weeks, during which his diet was restricted, with a view to avert the evils attendant on inflammation. Splints were then applied, and the leg "put up;" but the muscles had retracted, the bones overlapped, the foot came in contact with the ground on its outer edge only, and the limb was deficient in length to the extent of upwards of two inches.

The boy came under my observation at the expiration of thirteen months from the date of the accident, at which time he was unable to throw the slightest weight on the affected side. It did not appear, on careful examination, that the union of the bones was perfectly firm, and, having had the boy brought under the influence of chloroform, I separated the uniting medium, although not without difficulty. Extension by means of pulleys was made for three quarters of an hour. The distortion of the limb became greatly reduced, although still retracted in length to the extent of one inch. Splints were applied with more than ordinary care, the pressure of which produced extensive ulceration of the leg below the knee and about each ankle, and thus three weeks' time was lost. I then divided the tendo Achillis, and renewed the attempt to extend the leg by means of pulleys. This object was accomplished with some success. The natural form of the limb was restored, the bones united firmly, and the boy recovered the entire use of the foot. He places it fearlessly on the ground, and walks with an almost imperceptible limp, consequent on a retraction of the limb that does not exceed three quarters of an inch.

CASES OF DISLOCATION

OF THE

OS CALCIS AND SCAPHOID

FROM THE

ASTRAGALUS;

WITH

REMARKS ON THE IMPORTANCE OF DIVIDING THE GASTROCNEMIUS AND OTHER TENDONS,

TO FACILITATE REDUCTION IN VARIOUS DISLOCATIONS OF THE LATTER BONE.

BY

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H. N—, æt. 43, a large, stout man, was admitted into St. George's Hospital, under the care of Mr. Keate, in August, 1843.

The man had thrown himself out of a chaise, while his horse was running away, but was unable to give any accurate description of the manner in which he fell, or in what way his foot was injured. The right foot was found to be considerably bruised about the ankle-joint; the sole was somewhat everted; and on the inner part of the instep there was projecting inwards a prominent mass of bone, apparently the head of the astragalus dislocated

inwards and forwards. The integuments over this were tightly stretched and very much bruised.

From the amount of tumefaction of the soft parts, and the extensive bruising of the skin, it was somewhat difficult to determine the exact nature of the injury. The foot was fixed, and very little flexion could be produced. All movements gave rise to great pain.

Mr. Keate saw the man soon after his admission, and made great efforts to extend the foot from the leg, with the hope of reducing the dislocation. Not the slightest alteration was produced in the relative position of the displaced bones by these means.

Attempts at reduction were again repeated on several successive days, but each attempt entirely failed. It was then determined to leave the injured limb without further molestation. Pulleys were not used, as the soft parts appeared too much bruised to be able to bear any severe amount of pressure.

The integuments, which were tightly stretched over the prominent and displaced astragalus, soon sloughed; and within a short time a considerable external opening communicated with the tarsal joints. Suppurative inflammation subsequently extended up the leg; and, considering the amount of local mischief, and the accompanying constitutional disturbance, it was decided to remove the leg by amputation below the knee.

On examination of the foot, after its removal, the following conditions presented themselves. The astragalus was dislocated forwards and inwards from the os calcis and scaphoid. The upper surface of the astragalus was not dislocated from the tibia and fibula. The extremity of the external malleolus was broken off, but attached by the external lateral ligament to the os calcis. The deltoid ligament maintained the connexions between the tibia and astragalus. The head of the astragalus was lying internal to the anterior tibial tendon, on the upper and inner surface of the scaphoid; while the posterior inferior extremity of the astragalus was pressed into the groove on the upper

surface of the os calcis; the lower articular surface of the astragalus was in apposition with the posterior surface of the scaphoid. The sub-astragaloid (interosseous) ligament was torn through. The surfaces of the articular cartilages of the tibio-tarsal and larger tarsal joints were partially absorbed. The ankle-joint was open in front and behind, but it was uncertain whether this condition was not entirely the result of suppurative action subsequent to the dislocation.

The man died on the 7th of October.

R. L—, æt. 43, was admitted into St. George's Hospital in August 1858, in a state of delirium tremens. He was much bruised about the body; and there were two scalp wounds on the left side. An extensive lacerated wound occupied the back part of the left leg, extending through the integuments to the fascia, and from the lower part of the popliteal space half-way to the ankle. The left foot was much distorted and painful, and attracted immediate attention. There was some slight bruising of the skin, and superficial abrasions over the outer surface of the instep. A considerable prominence of bone presented itself on the outer side of the foot, in front of the external malleolus: the integuments were stretched tightly over the projection. The prominence was caused, apparently, by the head of the astragalus, this bone being dislocated from the articular surfaces of the scaphoid and os calcis. The foot was inverted, so much so, that its outer border was turned downwards, and the sole inwards. The internal malleolus could not be detected, being evidently buried in the soft parts of the inner side of the inturned foot. The external malleolus was very prominent, and its extremity on a level with the outer border, and now lowest margin of the foot. The tendo Achillis was very prominent, tense, and slightly curved inwards. The surface of the dorsum of the foot was shortened.

Mr. Venning, the house-surgeon, finding himself unable to reduce the dislocation, sent for me about 11 p.m. The

patient was then rather faint from loss of blood, and restless and talkative.

The account given of the accident was, that, in a fit of delirium, he threw himself out of a back window on the second floor of the Gloucester Coffee House, in Piccadilly, and that he had fallen through a skylight into a kitchen beneath. He was immediately carried to the hospital, and was seen by me within two hours of the accident.

It was at once apparent that the astragalus was dislocated outwards; the os calcis and other bones of the foot being thrown inwards. As the man was faint, and offered no resistance, the limb was at once flexed at the knee, and extension made on the foot. I grasped the heel and anterior portion of the foot, while Mr. Venning and Mr. Ash, the dresser, kept up counter-extension. Though extension was thus maintained, with as much power as could be safely applied, there was not the slightest alteration produced in the relative position of the displaced bones.

Recalling to my recollection the numerous unsuccessful attempts at reduction, made by Mr. Keate, in the case first related; and the amount of force which was uselessly applied in that instance; and recollecting the mischief which then followed, from the sloughing of the stretched and bruised integuments over the displaced astragalus, in consequence of relief not being afforded, either by reduction or otherwise, and feeling satisfied that, in this instance, violent efforts at reduction might be fatal to the already bruised integuments, and that delay in the reduction would only complicate this evil; I at once decided to divide the gastrocnemius tendon.

As the patient was partly unconscious, the division was made without placing him under the influence of chloroform. The wound having been dressed, extension was again tried; and, to our great satisfaction, the reduction of the bone took place immediately, and without the slightest difficulty. A splint was applied to the outside of the leg, which was allowed to lie on a pillow, so as to avoid pressure on the wound at the back part.

On the following day, the foot, though swollen, and having considerable subcutaneous extravasation of blood around it, presented no indications of unhealthy inflammation, nor any tendency of the integuments to slough. The man was still delirious, but more quiet than on the previous evening.

Subsequently, he became worse with general symptoms, independent of the condition of the limb. Into further particulars it is not necessary to enter, as the condition of the foot remained perfectly satisfactory until his death, which took place on the fourth day.

Post-mortem examination.—Two scalp wounds on the left side exposed the bone to a small extent. Extensive œdema of the scalp surrounded these wounds. There was a slight bruise of the surface of each posterior lobe of the brain, and corresponding to the bruises were superficial extravasations of blood into the arachnoid. The brain was very wet, and the lateral ventricles much distended with serum.

The tendo Achillis of the left foot was divided about one inch and a half above its insertion, and the divided portions were separated by an interval of one inch and five eighths. There was no kind of union between them. The bones of the foot were in their natural position, except that the rounded head of the astragalus projected outwards rather more than usual. The slightest force, however, was sufficient to displace the os calcis and scaphoid from the astragalus, so as to allow the foot to turn inwards, with the sole vertical. It was then seen that the interosseous ligament, between the astragalus and os calcis, was stretched, but not ruptured. The fibrous structures surrounding these two bones on the outside were torn, except a small slip, which was left, corresponding to the neck of the astragalus. The cartilage on the posterior articulating surface of the os calcis was slightly bruised. The external lateral ligament of the ankle was torn away from the bone of the leg, with the exception of the posterior slip. The internal lateral ligament remained entire. A small fragment of the astragalus at its posterior, internal, and inferior margin, and

the tip of the os calcis, with which the above portion of the astragalus articulates, were broken off. The astragalus was also bruised in a few places. The malleoli remained entire, except that there was a slight roughness, corresponding to the part from which the lateral ligament had been torn away. The ankle-joint was entire. There was a good deal of ecchymosis both in the dorsum and the sole of the foot, but no other injury of importance.

It will be observed that the injury, in both of the foregoing cases, was simply dislocation of the os calcis and scaphoid from the astragalus; the head of the astragalus, in the last case, having been thrust *outwards*, external to and below the extensor tendons, but the bone itself being in no degree dislocated from the tibia and fibula: in the first case, having been pushed *inwards*, but the ankle-joint being equally free, as in the last case. In fact, they were dislocations of the foot on the astragalus, or, rather, as Mons. Broca, in an excellent article addressed to the Society of Surgery in Paris, terms them, "luxations sous-astragaliennes."

It is not my intention to enter into a consideration of the variety of dislocations affecting the astragalus, such as these cases might suggest, further than is necessary to illustrate the advantages of the treatment advocated in this communication, and such as I adopted in the case last mentioned.

Every surgeon who has had any experience in dislocations common to the astragalus, whether the astragalus be simply thrust out from its box, *i. e.* its upper and lower articular receptacles; whether the astragalus be dislocated backwards; whether, as in the above cases, it is dislocated inwards or outwards on the os calcis and scaphoid; or lastly, whether the dislocation be complicated with an external wound; every surgeon will allow that attempts at reduction are generally attended with much trouble, often necessitate much violence, and are oftener unsuccessful; that, in most instances, the condition of the soft parts

is such, that the slightest attempts at reduction render these parts still more liable to slough than they may already be from the accident; and that, when these attempts are unsuccessful, they materially assist in hastening the destruction of the integuments.

I should needlessly occupy the attention of the Society, were I to dwell on the numerous recorded cases of dislocations of the astragalus, to illustrate the observations contained in this paper, or if I endeavoured to impress upon the members the very serious character of these accidents. At the conclusion of these remarks, a short table of cases will be found, which proves sufficiently how anxiously the surgeon should always regard the treatment of dislocations of the astragalus, and how serious may be the injury to the patient, when attempts at reduction are unsuccessful, and nothing worse remains to mark our defective skill than a permanently deformed foot.

The first case related in this paper occurred some fifteen years ago, and was the first case of dislocation of the astragalus I had ever witnessed. It happened when I was house-surgeon at St. George's Hospital, and I had every opportunity of watching its progress. The treatment and termination of the case impressed me strongly at the time with the following ideas. First, that, considering the anatomy of the tibio-tarsal and tarsal articulations, it was evident that in most varieties of dislocations of the astragalus, division of the larger posterior tendons of the leg must materially diminish the difficulties encountered in the attempts at reduction, if those difficulties would not be entirely removed by that division. Secondly, if division of the tendons did not prove beneficial to such an extent, and if the integuments over the displaced and prominent bone were much bruised and stretched, and, consequently, threatened to slough, it would be the best practice at once to liberate the integuments by a free incision; and that the astragalus should be removed as soon as practicable, so as to restore the foot, as nearly as possible, to its natural position.

I fully determined to put in practice the first of these suggestions, whenever it should fall to my lot to treat a case of dislocation of the astragalus which withstood ordinary efforts at reduction, in whatever direction the dislocation might occur. I had to wait until the present year before such a case came under my notice; and I have ample reason, I think, to be satisfied with the results of the treatment.

Dislocations of the os calcis and scaphoid, inwards or outwards, from the astragalus, may occur to such a partial extent, or the muscles of the leg may be so little resistant in some more complete cases, that reduction can be effected without much force, and without loss of time. A man was admitted into St. George's Hospital in the autumn of 1857, having fallen off a scaffold about twelve feet from the ground. He was taken into the ward while I was speaking to another patient in the same room. I was requested by Mr. Percival, the house-surgeon, to examine the man's foot. The left foot was slightly inverted; the head of the astragalus slightly prominent on the outside of the instep; the skin over it was not bruised, nor much stretched. The man stated that his foot bent under him when he came to the ground. The limb was immediately flexed at the knee, and extension made by my grasping the foot and heel, and drawing directly downwards. In a few seconds the reduction followed, attended by a peculiar sound, as if ligaments were being torn through, by the forcible separation of bones at a joint. In three weeks the patient was able to leave the hospital.

Mr. Paget has kindly favoured me with notes of the following case, which occurred in his practice at St. Bartholomew's Hospital.

A labourer, about forty years old, fell from a great height, and his left foot bent inwards beneath his weight. He was quickly taken to the hospital. There was no swelling about the foot or ankle, and the nature of the

injury he had received was as evident as it would have been in a skeleton. The astragalus was felt in its normal relation to the tibia and fibula, with the integuments stretched tightly over it. The foot was carried completely inwards; its axis being at right angles with that of the leg, and its sole flat, turned neither inwards nor outwards. It was a little drawn up towards the leg; the inner malleolus probably resting in the cavity in which the astragalus naturally rested.

The reduction of the displacement was easy. The knee being bent, the foot was pulled straight downwards, with one hand grasping the metatarsus, and the other the heel; and, as it yielded a little, the foot was pushed outwards. It at once slipped under the astragalus, with a distinct rub of the articular cartilages. There was no fracture of either tibia or fibula, and both malleoli were perfect. The patient had no hindrance to his recovery.

Such cases as these require very little consideration; they are attended with few difficulties, and they recover without deformity or much loss of time.

But dislocations of a more severe character are not so easily managed. Mr. Keate's case is a good instance of a difficulty in practice; though I believe that, if the gastrocnemius tendon had been divided immediately after the admission of the patient into the hospital, reduction would, in that case, have been readily effected, and would probably have been followed by much less mischief in the soft parts than subsequently supervened. In the preparation[1] may be observed the altered position of the astragalus. The preparation also explains the manner in which the efforts at reduction may be frustrated by the position and action of the tendons and their muscles.

If other cases were wanting to prove how difficult the reduction may be in similar instances, we can find them

[1] See preparation A. a. 87, Catalogue of St. George's Hospital Pathological Museum.

recorded in numbers. Mr. Arnott[1] reports a case in which the os calcis and scaphoid were dislocated outwards from the astragalus. The latter bone projected inwards, and there was a large wound exposing it. "The knee was bent, and counter-extension made. At first it was attempted to replace the bone, by grasping the heel and instep, then drawing the foot powerfully downwards, and at the same time endeavouring to press the astragalus back into its place by the points of the thumbs. No perceptible change was produced in the relations of the bones. A folded towel, previously wetted, was then carried round the outside of the foot, and its ends twisted together on the inside. By these means the foot was grasped very securely. Assistants were required to pull upon the foot, while Mr. Shaw endeavoured to guide the astragalus into its place, at the same time raising up the outer edge of the foot, with the view of depressing the inner border of the os calcis, to bring it under the level of the displaced portion of the astragalus. But these attempts proved equally unavailing as the first. It was next thought that a better direction might be obtained for the force, by twisting the towel on the contrary side of the foot, and then drawing upon it; but no advantage was gained by this method. In whatever way the force was applied, the astragalus remained immovably fixed. As the patient had undergone much severe pain, and as no hopes were entertained of reducing the bones, and as it was thought that to continue the attempts at reduction any longer would only be adding to the severity of the injury already received, it was determined to desist from making any further trials." The same evening the astragalus was removed by Mr. Shaw, and the patient subsequently recovered with a useful limb.

This was a case of *compound* dislocation. Some surgeons, as I shall hereafter mention more particularly, consider reduction, in such a complication, next to impossible. But

[1] 'London Medical Gazette,' vol. xx, p. 588.

if the amount of force necessary to successful reduction in some cases of *simple* dislocation, is to be fully appreciated, I cannot do better than notice shortly a case which occurred to Mr. Hancock in Charing Cross Hospital. The os calcis and scaphoid, in that case, were dislocated outwards upon the astragalus; and there was no wound of the skin. "I endeavoured," says Mr. Hancock, "to reduce the dislocation by placing the foot on its outer edge upon my knee, pressing it up, while I, at the same time, drew the front of the foot downwards and forwards with my hands, but unsuccessfully; therefore, under the circumstances, and fearing that if something were not done to relieve the tension, extensive sloughing of the integuments would ensue, I determined to apply the pulleys; but I did not make any very great efforts, as I found it necessary to have an apparatus made on purpose, and was unwilling to produce more disturbance than was absolutely necessary."[1] An apparatus, which he describes, "was employed to make extension; it was kept up for one hour and a quarter;" after which time "the reduction was effected," seven days after the occurrence of the dislocation. The integuments over the instep subsequently sloughed, and exposed the bone. About three quarters of an inch of the astragalus was afterwards removed; and in seven months the man left the hospital with a useful limb.

The few cases of dislocations of the astragalus from the os calcis and scaphoid, mentioned by Sir A. Cooper, furnish excellent evidence of the difficulties met with in attempts at reduction. "In these cases," he says, "the use of pulleys will be required, and the action of the muscles should be lessened by tartarised antimony." In fourteen cases of compound and simple dislocation recorded in his work, reduction, by ordinary measures, was effected in three only. "The difficulties of reduction," he says, "seemed to arise from the small size of the wound in the capsule of the joint, and in consequence of the bone being tightly held

[1] 'Lancet,' vol. ii, p. 36, 1844.

by the tendons." In speaking of dislocation of the tibia inwards from the astragalus, "great force is required," he says, "if the limb be placed in the extended position, from the resistance of the gastrocnemius."[1] Notwithstanding that these remarks point so directly to the action of the gastrocnemius and its tendon, and to the necessity of relaxing, or dividing the latter, a case is recorded by him, of dislocation of the tibia forwards, in which unsuccessful attempts with simple extension alone were made, and the deformity remained for life. The dissection of another case is also described by him, accompanied by an engraving of the displacement, which indicates clearly that the action of the gastrocnemius interfered chiefly with the reduction.

"Dislocations of the astragalus," says Mr. Turner, "are accidents of unfrequent occurrence, in comparison with many others which happen to the human body. But in looking over the list of surgical accidents, there is not one more serious in its character and in its consequences; and there is not one in which we are so destitute of rules to guide us in our practical proceedings. What are the obstacles to reduction in most cases?" he asks, and "since reduction is impracticable, in almost all cases of dislocation of the astragalus, by what principles are we to be governed in the treatment of this accident?"[2] In reply, I trust I shall be able to establish,

1st. That certain principles should guide us in our practice.

2d. What the chief obstacles to reduction are.

3d. That reduction is not impracticable in the great majority of cases.

A case in most respects similar to that of R. L—, the second related in this paper, is reported by Dupuytren.[3] The comparative results of the treatment, in each case,

[1] 'Treatise on Dislocations and Fractures.'

[2] 'On Dislocations of the Astragalus,' by Thomas Turner, Esq., Surgeon to the Manchester Royal Infirmary.

[3] 'Injuries and Diseases of Bones.' Published by the Sydenham Society.

are practically important, and deserve our attention. A man fell down as he was going into his house; he immediately felt an acute pain in his left foot, and perceived that it was deformed. He was taken to the Hôtel Dieu, on the following morning. The foot was found twisted considerably inwards; the point of the internal malleolus could not be felt; the outer malleolus was prominent; there was a very distinct prominence of a rounded form, anterior to the outer ankle. The swelling was moderate, and the foot appeared a little shortened. The tibia and fibula were sound. Extension was made in a most energetic manner several times, but as these efforts did not succeed in reducing the dislocation, the patient was bled and kept in a tepid bath for some hours. On the following day renewed attempts were made at reduction, but they only proved so far effective, that though the dislocation was not entirely reduced, there was a manifest improvement in the shape of the limb. A splint was applied to the outer side of the leg, so as to give the foot a more natural direction. The man would not submit to anything else being done to the limb; and when he left the hospital, the foot was only slightly inverted, and the points of the toes directed a little more downwards than natural.

A case, in many respects similar, is related by Mr. Turner, as having occurred to Mr. Smith, of Leeds. Attempts at reduction were made for two hours without success; after which, a second trial was made to reduce the dislocation by means of pulleys; this also failed. Subsequent sloughing of the integuments took place, and the astragalus was removed at a later period, the patient recovering with a useful limb.

It must not be supposed that I claim credit to myself, as the first to suggest the division of tendons in the leg to facilitate reduction of dislocations of the astragalus; nor am I the first to have divided these tendons in order to obtain such a result. Mr. Turner alludes to the practicability of this operation in the following terms: "tenotomy may be regarded as a means of facilitating reduction, and in this

point of view the practice is justifiable." I should state that I had not seen these observations when I first decided to put in practice the division of the tendo Achillis in cases of dislocations of the astragalus. I consider myself fortunate in having my suggestion strengthened by the support of such an authority in his profession, and more than satisfied that the practice, which so completely succeeded subsequently in my hands, had already been alluded to by a surgeon of such experience as Mr. Turner.

Mr. Fergusson, writing on dislocations of the astragalus, says, "it has been proposed in cases where the reduction is difficult, to divide some of the neighbouring tendons by subcutaneous incisions, and possibly in certain instances there might appear some encouragement to do so; certainly the practice would, if judiciously applied, do no harm."[1]

In volume xxxiii of the 'Transactions' of this Society, is published a paper by Mr. De Morgan, on the section of the tendo Achillis in certain cases of fracture of the bones of the leg. The proposal to divide the tendon in such cases is well worthy the attention of all surgeons, and confirms, in a great measure, the view taken in the present communication, of the importance of dividing the tendon in dislocations of the astragalus, whenever the slightest difficulty is experienced in an attempt at reduction. It may be supposed that the action of the gastrocnemius is much more evident, and direct, and powerful in *producing* displacement in an oblique fracture of the tibia and fibula, than in *maintaining* displacement, when opposing our efforts at reduction in a case of dislocation of the astragalus. But I can only urge in argument, that the manifest facility with which the reduction took place, after division of the tendon in the case already related by me, not only strongly confirms the principle of Mr. De Morgan's proposal and practice, but also establishes something like a principle in the treatment of all cases of dislocation of the astragalus.

In the first volume of the third series of 'Guy's Hospital

[1] 'System of Practical Surgery,' third edition, p. 364.

Reports,' will be found an interesting account of several cases of fracture of the leg, and dislocation of the ankle-joint, which occurred respectively under the care of Mr. Cock, Mr. Birkett, and Mr. Poland, and in all of which the tendo Achillis was divided. These cases, in every respect, confirm Mr. De Morgan's views respecting the great advantages derived from dividing the tendon in certain injuries of the leg, and they also in most respects bear upon my proposal. One case, however, requires especial consideration: it is reported as "one of well-marked dislocation of the astragalus." Mr. Birkett, under whose care this patient was, has kindly furnished me with some further particulars of the dislocation and result of treatment, which will be referred to presently, as they bear on another point of treatment in certain forms of dislocation.

As a general rule, and as *the* principle to guide us in our practice in dislocations of the astragalus, I do not hesitate to recommend the division of the gastrocnemius tendon, in all cases in which moderate extension with the hands of the surgeon does not succeed. I trust I may not be considered too sanguine in venturing to express a belief, that *most* cases of dislocation will thus be successfully treated.

I purposely make use of the guarded term, that *most* of these dislocations can be reduced by the adoption of the treatment now advocated. The question naturally arises, will not all cases be equally benefited by its adoption? I think not. I hope I shall prove satisfactorily, that in some few exceptions, though the gastrocnemius tendon should be divided as a preliminary measure, if that be not sufficient, the division of the tibialis posticus tendon will be necessary, and will be sufficient to allow the ready reduction of the displaced bone.

The most common forms of dislocation of the astragalus, as far as I have been able to ascertain, appear to be those either outwards or inwards on the os calcis and scaphoid, in which the astragalus is not dislocated at the same time from the tibio-fibular surface. Mons. Broca appears also to

be of this opinion. Dislocations of the astragalus, complete from all its corresponding articular surfaces and attachments, I believe to be the most rare of all dislocations implicating this bone. Now of the dislocations *outwards*, those that cannot be reduced in the first instance by manual extension, I believe, will be always under manual control, *subsequent* to division of the tendo Achillis. My own case gives evidence in favour of this assertion. In this form of dislocation the external tendons rarely appear to be implicated in any way with the displaced bone. I cannot find the record of a single instance in which such a complication existed. But when the astragalus is dislocated *inwards*, the os calcis and scaphoid being thrown outwards, then it sometimes happens that the tendon of the tibialis posticus is thrown in front of the neck of the astragalus, and so closely embraces it, that the head of the bone is fixed between the tendon and the calcaneo-scaphoid ligament to such an extent that reduction is impossible until this tendon be divided, either alone or in conjunction with the tendon of the gastrocnemius. It will be seen at once, that if the above condition respecting the tibialis posticus tendon prevails, extension only aggravates the obstruction, by tightening the very tendon that holds in its embrace the constricted neck of the astragalus, the head of which has to be drawn inwards between this tendon and the ligament beneath.

The following cases illustrate this displacement of the tendon, and, I think, justify me in drawing the foregoing conclusions.

The first case is reported anonymously in the 'Clinique de Marseilles,' and is also mentioned by Mons. Broca. A man, 63 years of age, was taken to the Hôtel Dieu, at Marseilles, in October, 1842. He had fallen from some height, and had a severe wound on the left foot. The foot was everted almost transversely, the toes pointing outwards, and the heel inwards; the sole of the foot was not unnaturally placed. The inner margin of the foot presented a prominent angle, the summit of which was formed by the

head of the astragalus. This was exposed, and projected through a wound, which extended from the dorsum of the foot, passed below the internal malleolus, and terminated near the tendo Achillis. The neck of the astragalus was entangled between the tendon of the tibialis posticus, which embraced its upper half, and the inferior calcaneo-scaphoid ligament, which embraced its lower half. The scaphoid was situated external to and a little above the head of the astragalus. The posterior tibial vessels and nerves were exposed in the wound; they were much stretched, but not lacerated. There was a comminuted fracture of the external malleolus. The reduction was not possible until after the division of the tendon of the tibialis posticus. The reduction then became easy, though very painful to the patient. He died on the fifth day.

Post-mortem examination.—The astragalus was in its place; the os calcis, scaphoid, and cuboid presented their natural relations, and their ligaments were intact. The ligaments which united the astragalus to the bones of the leg were not torn, but the sub-astragaloid (interosseous) ligament was ruptured for the greater portion of its extent. The external fibres only remained.

I am indebted to my friend Mr. Birkett for the following case.

C. H—, æt. 45, was admitted into Guy's Hospital in August, 1853. There was well marked dislocation of the astragalus inwards, from the os calcis and scaphoid. The foot was everted, and a prominence existed in front of the internal malleolus; the integuments were much stretched over the projection, but there was no wound of the skin. Extension was made with the hope of reducing the dislocation, but entirely failed to produce any effect on the displaced bones. On the following day the tendon of the gastrocnemius was divided by Mr. Birkett, and extension again made, but with no better success than before the division of the tendon. As sloughing of the integuments subsequently occurred, Mr. Birkett cut down upon the

astragalus in order to remove it, but he experienced some difficulty in endeavouring to do so, in consequence of the tendon of the tibialis posticus lying over, and in front of the neck of the bone, which was consequently tightly fixed in its new position. As soon as the tendon was divided, the bone was readily removed. Subsequent amputation of the leg was performed, and the patient ultimately recovered.

Mr. Poland was kind enough to favour me with the particulars of the next case.

A young man was admitted into Guy's Hospital with dislocation of the astragalus inwards: all attempts at reduction proved ineffectual. There was no wound in the skin, but subsequent sloughing of the integuments exposed the dislocated bone. The patient did not survive the injury many days. On examination of the foot the tendon of the tibialis posticus was found running in front of the neck of the astragalus, and, as in the former cases just related, no doubt prevented or assisted in preventing the reduction of the bones, when extension was made.

In the 2d volume of the 'Bulletin de la Faculté et de la Société de Médecine,'[1] a case is mentioned of dislocation of the astragalus, in which all attempts at reduction failed until the tendons of the tibialis anticus and posticus muscles had been divided. There are on record three cases of dislocation of the astragalus backwards; reduction was not effected in any one instance. It appears to me that division of the tendo Achillis would prove all important in any future instance of this rare injury.

It appears doubtful, at present, how far it may be desirable to replace the astragalus in cases of compound dislocation of this bone, if the attachments of the bone have been much destroyed. In compound dislocations the safety of returning the astragalus is, in Mr. Turner's opinion, a "very serious consideration, since if the bone be so loose and detached as

[1] P. 245.

to be capable of being easily reduced, is it not likely," he asks, "that it will die from want of sufficient vascular connexions?" It will, I trust, be fully understood that I have no desire to urge the division of tendons in all extreme cases. Every surgeon can best judge in the individual instance, whether it may be desirable to reduce a compound dislocation by simple measures, and at once replace the bone in its natural position, and thus relieve the integuments of all tension and pressure; or whether it may be more expedient and safer to remove the bone at once, or even to have recourse to amputation. Mr. Turner is of opinion, that in preference to any attempts at reduction, "extirpation ought always to be performed" in compound dislocations; and he adds, that in the experience of the surgeons of the Manchester and Leeds Infirmaries, a case of complete compound dislocation of the astragalus had never been reduced, nor had he been able to ascertain that any reductions in similar injuries had been accomplished in the Liverpool, Bristol, or other provincial hospitals. "All attempts at reduction are not only hopeless, but prejudicial," he says; "but the discouragement does not arise from the non-success of the practice alone, but from the mischief which often results from torturing attempts at reduction."

Now, although the cases mentioned in Sir Astley Cooper's work disprove, to a certain extent, the correctness of Mr. Turner's conclusions respecting the treatment of compound dislocations of the astragalus, yet the last quoted paragraph indicates to my mind that surgeons may modify their opinions and practice in this interesting and important question, after mature consideration of the treatment advocated in this communication: at any rate, that this treatment may be adopted in many instances, before proceeding at once to the removal of the astragalus: and I am strengthened in this expectation, when reminded that in three cases of compound dislocation of the astragalus, mentioned by Cooper, reduction was effected in each, and each case recovered.

It would be presumption on my part to speak decidedly as to the line of practice to be pursued in every case of compound dislocation of the astragalus, but I should certainly be inclined to divide the tendo Achillis in almost all such cases. I believe that the reduction of the bone would generally be secured readily without forcible means, and that the soft parts would be subjected to much less disturbance than if the immediate removal of the bone were attempted. If, however, the bone were much shattered, its removal must no doubt be desirable; but if it were broken without much comminution, and there did not appear much necessity for its immediate removal, division of the tendon would, in my opinion, conduce much towards the quietude of the injured parts, nor would the operation itself involve important considerations.

M. Broca prefers attempting reduction in the first instance in compound dislocations of the astragalus. The relaxation of the soft parts, by incision if necessary, and the section of certain tendons, he says, should facilitate the reduction of the bones; but that, notwithstanding these measures, the dislocation will often remain unreduced. He further adds: "1st. In dislocations *without wounds* it is necessary above all means to try reduction; if this is not effected it behoves us to wait. If abscess or sloughing follows, subsequent extraction of the astragalus must be performed. 2dly. In dislocation *with wounds* it is also necessary to attempt reduction; and in having recourse to it, if necessary, to relieve the skin, and also to have recourse to tenotomy."[1]

It is interesting to observe how near Mons. Broca has approached the true appreciation of the importance of dividing tendons in dislocations of the astragalus without having thoroughly established, as a rule, their division in every such case. He does not allude to their division in the treatment of simple dislocations, in which it will, no doubt, prove most beneficial, although he recommends

[1] 'Mémoires de la Société de Chirurgie de Paris,' vol. iii.

tenotomy generally in compound dislocations, in which cases it may sometimes be impracticable, from the severity of the injury. M. Broca does not, however, appear ever to have practised division of any tendons in any form of dislocation of the astragalus.

I may add in concluding the consideration of compound dislocations in connexion with division of the tendons, that Dupuytren's opinion, as regards the treatment of such cases, was that "the removal of the astragalus was far preferable to repeated attempts at reduction."

The figures in the appended table point out most strongly the seriously damaging character of dislocations of the astragalus, even under some apparently favourable circumstances, unless reduction be effected with gentle means. I trust, therefore, I am not too urgent in my endeavour to impress upon the Society and the profession generally, the advantages of dividing the gastrocnemius and other tendons in all dislocations of this bone, if simple extension by ordinary measures does not succeed; and that rather we should have recourse to it in all cases, in order that we may, as far as practicable, avoid contusion of parts already bruised, and often severely lacerated; and that we may put in practice this treatment, as a far preferable preliminary to, if not an entire substitute for pulleys. Will any surgeon dispute the advantages of dispensing with pulleys, at the expense of the division of a tendon, when he calls to mind the extensive mischief that has followed even their successful application?

Extraction of the astragalus is an operation, frequently of remarkable innocence, as far as life is concerned. But I am clear that it would have been much less frequently practised if reduction could have been readily effected; and that it has only been performed, when extension failed, rather than leave the bone prominent, and pressing on the stretched and contused integuments.

The following appear to me the most reasonable rules to guide us in our practice:

1st. In all *simple* dislocations of the astragalus *outwards*,

from the os calcis and scaphoid, in which moderate extension fails to effect reduction, the gastrocnemius tendon should be divided, and extension subsequently made.

2dly. In all *simple* dislocations of the astragalus *inwards*, if moderate extension fails, the gastrocnemius tendon should be divided; if this be not sufficient to permit reduction under gentle extension, the division of the tibialis posticus tendon should be had recourse to, for reasons already mentioned. Some care may be required to divide this tendon, as it is sometimes displaced from the groove behind the internal malleolus.

3dly. In *compound* dislocations, not reducible under gentle extension, division of the tendons, which interfere with the reduction, should at once be performed.

I must beg leave, in conclusion, to remark that too much must not be expected in these injuries from division of the tendons necessary to facilitating the reduction of the dislocation. I do anticipate that the operation will usually and materially assist reduction. I do not anticipate that it will, to any great extent, modify or affect the subsequent treatment or progress of severe cases. But I believe it will materially diminish the mischief in the soft parts which is liable to follow the severe handling in extension, or the pressure caused by the application of pulleys, or other measures in unsuccessful attempts at reduction. If, however, any satisfactory results are obtained by the adoption of such a simple and readily performed operation as division of the tendons mentioned, in but a small number of cases of this formidable accident, I trust that the members of the Society will not consider this communication on my part a useless one, but rather that the principles advocated in it are based on sound and practical considerations.

Results of treatment in fifty-five cases of Simple and Compound Dislocation of the Astragalus.

Nature of Dislocation.	Treatment.	Result.	Total.	
Simple dislocation, 32 cases.	Amputation	Recovered. 1 Died 3	4	In one case tendo Achillis divided.
	Extraction of astragalus	Recovered. 3 Died ..... 1 Uncertain. 1	5	
	Reduced ...	Recovered. 13 Died 1	14	In one case tendo Achillis divided.
	Not reduced	Recovered. 8 Died 1	9	
		Total of cases	32	Recovered. 25 Died ..... 6 Uncertain. 1 32 cases.
Compound dislocation, 23 cases.	Amputation	Recovered. 0 Died ..... 3 Uncertain. 2	5	
	Extraction .	Recovered. 11 Died 2	13	
	Reduced ...	Recovered. 2 Died 2	4	In one case tendon of tibialis posticus divided.
	Not reduced	Recovered. 1 Died 0	1	
		Total of cases	23	Recovered. 14 Died ..... 7 Uncertain. 2 23 cases.

The total number of deaths was 18 out of 55 cases of compound and simple dislocation of the astragalus. This rate gives an average of 1 death in $4\frac{1}{4}$ cases of dislocation.

The total number of reductions was 18 out of 55 cases. Three of these died; leaving 15 cases of recovery after reduction, treated by the usual methods of extension. These give an average of about one reduction in every four cases of dislocation of the astragalus.

A DESCRIPTION

OF THE

ORGANS OF GENERATION

OF A

HERMAPHRODITE SHEEP.

BY

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COMMUNICATED BY

F. C. SKEY, F.R.S.

Received Jan. 11th.—Read Jan. 25th, 1859.

I AM indebted to Mr. Paget for the opportunity of examining these organs.

During life this sheep was regarded as an ewe.

The whole of the external organs are not attached to the specimen; but, from the description given, there is no reason for doubting that they presented the characters of those of an ordinary female.

The external orifice leads into the vagina, beyond which is the uterus, with its two horns, as in the female.

In the place of the ovaries, each surmounted in its whole length by an epididymis and by the termination of the corresponding uterine horn, are two testes.

In shape and aspect these testes are precisely similar to those of an ordinary male, but very much smaller—perhaps not more than one fourth of the size of the fully developed male organ. A section presents to the naked eye the ordinary appearance. Its minute structure is also that of a natural testis (*see* Plate II). The only important difference which a careful examination detects, is the absence of any traces of spermatozoa.

On either side of the vagina is a well-formed seminal vesicle, which opens, in common with a tube to be presently described, into its anterior wall. The urethra, from the bladder in front, after running for some distance in the substance of the anterior wall of the vagina, opens into it, between one and two inches from the vulva.

With the exception of the testes, whose relations are precisely those of the ovaries, the epididymes, and the seminal vesicles, the organs differ from those of the ordinary female only in the following minute details.

In the female, when the uterine horn reaches the ovary, it suddenly tapers to a point, from which a fine, convoluted Fallopian tube, about two inches long, is continued over the ovary, but not in contact with it; only connected to it by the peritoneal fold—the broad ligament.

In this specimen the uterine horn terminates, as usual, in a fine tube, much longer than natural, which commences in connexion with the epididymis at the inner extremity of the testis. It is continued, bound closely to the epididymis, over the whole length of the testis from the inner to the outer extremity, connected to it by a fold of the tunica vaginalis, and passing beyond its outer extremity for more than two inches, it turns back upon itself, and, suddenly enlarging, passes over the testis a second time, from without inwards, as the horn of the uterus. Thus the slender terminal portion of the uterine horn has the same relation to the epididymis as the commencement of the vas deferens holds to that body in the male.

The body which surmounts the testicle, in shape and

relation to that organ corresponds exactly to the epididymis. It, however, exhibits no structure beyond that of condensed areolar tissue. It is connected at its inner extremity with the termination of the uterine horn before noticed, and from its outer extremity—the thickest portion—proceeds a delicate cord or tube, which, closely connected with the under surface of the uterine horn, passes down to the fundus of the uterus. When it reaches that organ it suddenly increases considerably in size, and passes through its anterior wall, and that of the vagina, parallel with its fellow from the opposite side, as a well-developed tube; and finally opens in common with the corresponding seminal vesicle.

Where the epididymis commences, at the inner extremity of the testis, a process, about an inch and a half long, springs from the testis like a commencing epididymis, and is composed of imperfectly developed convoluted tubes, imbedded in areolar tissue, but it terminates in a blind extremity; and the portion I have already described as the epididymis has no connexion whatever either with this process or with the testis, beyond being bound to them by cellular tissue and membrane.

The uterus is altogether smaller than that of the female; it measures two inches in length instead of three, and its walls are thinner, especially in the lower half of its body, the walls of which, in the female, are here very firm and coarsely rugous. The rugæ of this uterus are much finer and more delicate than those of the ordinary female; but the distinction between uterus and vagina is most strongly marked and indicated by a well-developed os.

The horns of this uterus are more slender than in the female, and at least twice the length. The surface of the mucous membrane of the horns and upper portion of the uterus is smooth and uniform, and there is no appearance of those circular or oval glands which are so abundant in the mucous membrane of the horns of the female uterus, and which, from their prominence and the layer of black pigment capping each, are such remarkable objects.

The vagina is exactly half the length of that of the female; three inches instead of six.

The urethra, also, is half as long; one instead of two inches.

The seminal vesicles are each an inch and a half in length.

This case is remarkable for the complete union of such perfect male and female organs. It will be observed that it belongs to that class of hermaphrodites of which the first of the three free-martins described by Hunter, Mr. Wright's free-martin, is an example. In this case, however, "the vagina terminated in a blind end, a little way beyond the opening of the urethra, from which the vagina and uterus were impervious." Moreover, Hunter says of the bodies he calls testicles, "When cut into they had nothing of the structure of the testicle. Not being similar to anything in nature, they had more the appearance of disease;" and "there was nothing similar to the vasa deferentia." (Hunter's Works, vol. iv, pp. 41-2.) I will not trespass upon the attention of the Society by comparing or contrasting this case with other recorded examples which more or less resemble it, for the inquiry would be neither instructive nor interesting. But, for some of such cases, I would refer especially to Dr. Simpson's elaborate essay on Hermaphroditism, in the 'Cyclopædia of Anatomy and Physiology.'

Of course this animal was really a male. The knowledge we already possess of the early development of the organs of generation, of the changes they subsequently undergo, and the relation of the different organs of one sex to those of the other, will, I think, go far to explain the production of such a monster as this one. At the same time, it illustrates the original unity of type, and affords additional evidence of the true homology of the individual organs in the male and female.

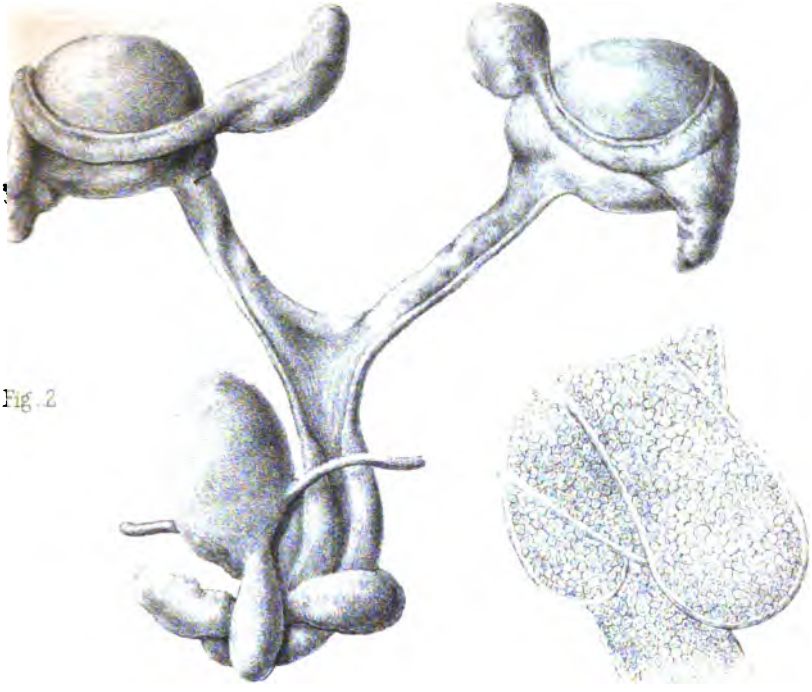

Fig. 2.

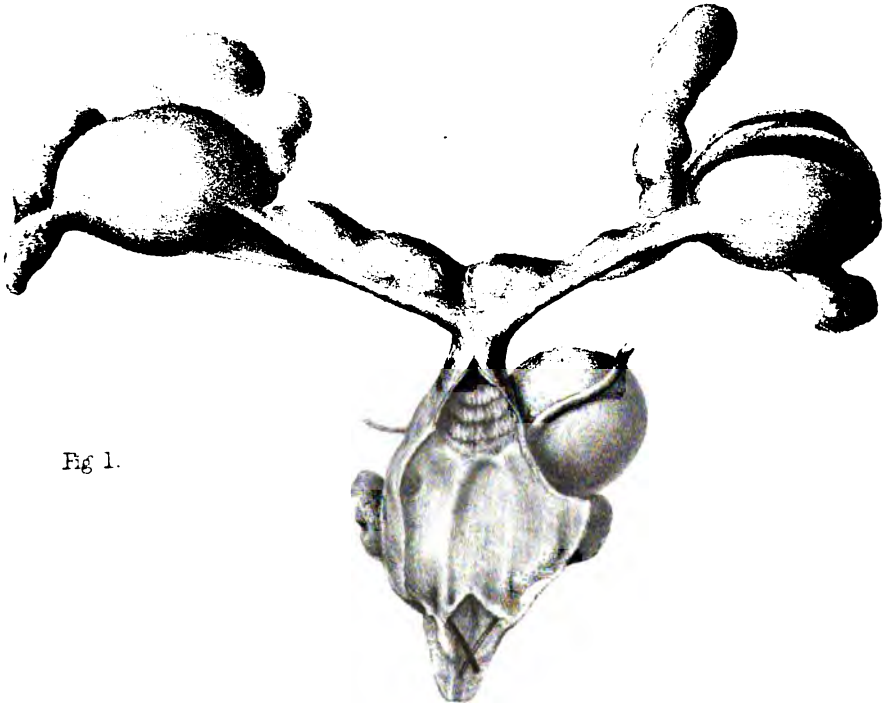

Fig 1.

ON THE

CAUSES OF DEATH AFTER AMPUTATION.

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INVESTIGATIONS into the causes of death have ever been sources of much interest to the members of our profession; and although, from the want of definite facts, this subject has always been more or less wrapped in obscurity, still, in these days, when men are fully aware of the fallacies and dangers which follow upon a practice based upon mere opinions or impressions, gained by experience however extensive, the value of definite facts becomes daily greater; and in this statistical age we are apt to test all our knowledge and opinions by numbers. Where such a test can be applied, no one doubts its value, for figures have now a recognised and undoubted authority in our science, as in others, and wonderful indeed are the results which they have produced.

It is true that we occasionally meet with men who dispute their power when applied to such a science as ours, yet these same men trust to what they call their experience.

What, then, is experience? Is it an indefinite something which is non-communicable to others, and is not to be expressed, or is it a result more or less definite, arrived at through practical knowledge?

If the latter, as I believe true experience to be, surely it is capable of expression, and, in a measure, becomes statistical; and, however limited it may be, it can be represented by a number, from which to argue, or upon which to base an opinion.

Experience, to be of value, must be something definite; it must depend upon something more than mere impressions, although these may be the result of extensive observation.

Knowing, then, how impressions only lead men astray, it is our duty always to define our experience by recording observations, and then, by a fair analysis of cases of a similar character, to bring out our results; for it is true that such results, if fairly obtained, are of more practical value than any impressions, however gained. But to do this, much care is needful; for by classing together or by comparing dissimilar cases, a wrong result must ensue, and discredit, with erroneous conclusions, will necessarily follow.

With a view of gaining some distinct idea of the causes of death after amputation, I have collected from Guy's Hospital 300 cases of amputation, and have divided them into four classes. The ordinary division of traumatic amputations into primary and secondary I have not thought necessary to alter; but when we come to what are generally described as pathological amputations, it becomes evident that in the classing together of such cases as amputations for talipes, tumours, elephantiasis, deformity, and others of a like character, with those for diseases of the joints, a wrong result must ensue; and practically this is found to be the case.

I have, therefore, divided these cases into pathological amputations and amputations of expediency, choosing this term as more accurately expressing the reason for the operation; since limbs are removed for tumours, talipes,

elephantiasis, and deformity more from expediency than necessity; and I would therefore suggest the use of such a term until a better is proposed.

The literature of this subject is very scanty indeed; with the exception of a valuable paper by Mr. James, published in the eighteenth volume of the 'Transactions of the Provincial Medical Association,' I am unable to point to any single memoir, in which the causes of death after amputation have been made a separate subject of investigation.

Mr. Alcock, in the twenty-third volume of the 'Medico-Chirurgical Transactions,' in a paper upon the Joints, made some few observations upon it, and promised at a future period to give the profession his views generally upon the causes of death after operations; but in this the Profession has been disappointed; and in presenting this short memoir to the consideration of my Surgical brethren, I trust that the imperfections of its pages will be counterbalanced by the importance and interest of the subject, and that others more competent to undertake such an investigation will follow my example, and in future fill up the deficiencies, which, I doubt not, are too numerous.

TABLE A.—*Of Amputations.*

	Pathological.			Amputations of Expediency.			Primary.			Secondary.			Total.			
	Cured.	Died.	Per Cent.	Cured.	Died.	Per Cent.	Cured.	Died.	Per Cent.	Cured.	Died.	Per Cent.	Total.	Cured.	Died.	Per Cent. and Average.
Thigh	82	18	18, or 1 case in 5·5	13	6	31·57, or 1 case in 3·16	8	12	60, or 1 case in 1·5	1	3	75, or 1 case in 1·3	143	104	39	27·27, or 1 case in 3·6.
Leg	36	3	7·7, or 1 case in 13	2	4	66·6, or 1 case in 1·5	9	15	62·5, or 1 case in 1·6	4	8	66·6, or 1 case in 1·5	81	51	30	37, or 1 case in 2·7.
Foot	5	...	...	...	...	...	1	...	...	...	...	...	6	6	...	...
Shoulder	...	...	...	1	...	...	2	...	...	...	...	...	3	3	...	...
Arm	10	...	...	3	...	...	10	3	23, or 1 case in 4·33	4	1	20, or 1 case in 5	31	27	4	12·9, or 1 case in 7·7.
Forearm	13	...	...	4	...	...	13	3	18·7, or 1 case in 5·3	3	...	...	36	33	3	8·3, or 1 case in 12.
Totals	146	21	12·57, or 1 case in 8	23	10	30·3, or 1 case in 3·3	43	33	43·4, or 1 case in 2·3	12	12	50, or 1 case in 2	300	224	76	25·3, or 1 case in 3·9.

Analysis of Table A.

Pathological amputations.—If we refer to the pathological amputations, as displayed in the first column, it will be seen that there are 167 cases, including

100	of the	thigh,	or about	60	per cent.
39	„	leg	„	23	„
5	„	foot	„	3	„
10	„	arm	„	6	„
13	„	forearm	„	8	„

Of these, 21 only were fatal, *i. e.*, 12·5 per cent., or about 1 case in 8; but as the fatality of amputations of the upper and lower extremity differs so widely, it is clearly incorrect to mix them together in statistical calculations, as by so doing we gain but an imperfect notion of the fatality of each.

By reference to the table it will be observed that out of the 100 cases of pathological amputations of the thigh, 18 were fatal, or 18 per cent., or 1 case in 5·5. Of the 39 cases of amputation of the leg, 3 only were fatal, or 7·7 per cent., or 1 in 13; and, taking pathological amputations of the thigh and leg together, 15 per cent. were fatal.

Amputations of the foot, arm, and forearm were all successful.

Amongst the 100 cases of amputations of the thigh, 89 are registered as being operated upon for chronic disease of the joint; and of these 13 proved fatal, or about 1 case in 7. (The fatality of excision of the knee-joint is, according to Butcher, at least 1 in 5.) Four were amputated for acute suppuration of the knee, and all died; 1 for gangrene of the leg, after ligature of the femoral artery for ruptured aneurism—also fatal. The remaining 6 all recovered; 4 being amputated for necrosed and carious bone,

1 after excision of the knee, and 1 for disease of the cellular membrane.[1]

Amputations of expediency.—If we pass on to the next column, containing the statistics of the amputations of expediency, a very different result will be manifested to that which we have just given. Of the whole number of 33 cases, 10 were fatal, or 30 per cent., or 1 in 3·3.

Amputations of the *thigh*, instead of yielding 18 per cent. of fatal cases, are there marked 31·57 per cent., or 1 in every 3·16 cases.

Amputations of the *leg*, instead of being 7·7 per cent. fatal, are registered as 66·6 per cent.

Amputations of the *lower extremity* as a whole are 40 per cent. fatal.

Those of the *arm* and *forearm* are all cases of recovery.

The difference in the fatality of the pathological amputations and of those of expediency, proves the error of associating them together in statistical or other calculations; and we now, by their separation, gain a more correct idea of the fatality of each.

The causes of death will be discussed as we proceed.

For what disease or cause amputated.—Twenty-nine of the 33 cases were amputated for simple or malignant tumours of bone, or elephantiasis; including 19 of the thigh, 6 of which were fatal; 8 of the leg, 2 of which were fatal; 1 of the shoulder, 2 of the arm, and 4 of the forearm, all of which recovered.

Two amputations for talipes were fatal.

One after a Chopart, and a second after a compound fracture for deformity, recovered.

Taking the amputations of the lower extremities for tumours alone, 36 per cent. were fatal; of the upper extremity all recovered.

[1] In the original two cases of amputation for conical stumps were included, but it has been thought desirable to omit them, and to add the next two cases from my book of amputations.

Traumatic amputations.—When we analyse the columns containing the statistics of the traumatic amputations, it will be seen that nearly 50 per cent. of the whole number proved fatal, and that there is but a slight difference between primary and secondary amputations; that difference, however, being in favour of the primary.

Taking amputations of the *thigh*, 60 per cent. of the primary were fatal, and 75 of the secondary; of the *leg*, 62·5 per cent. of the primary were fatal, and 66·66 per cent. of the secondary.

Taking both together, of the primary, 61 per cent. were fatal; of the secondary, 68 per cent.; the difference in favour of the primary being 7 per cent.

Amputations of the upper extremity in this division become more fatal, 7 cases proving fatal out of 39, or 18 per cent.

The causes will be alluded to in another page.

Analysis of Table A *as a whole.*

In considering amputations of the thigh as a whole, it will be seen that there are 143 cases; of these 39 were fatal, or 27·27 per cent., or 1 in 3·6. The order of fatality is as follows:

	Fatal.
1.—Pathological amputations .	18· per cent., or 1 in 5·5
2.—Amputations of expediency	31·5 „ or 1 in 3·16
3.—Primary amputations	60· „ or 1 in 1·5
4.—Secondary „	75· „ or 1 in 1·33

Amputations of the *leg* as a whole were fatal to the extent of 37 per cent., or 1 case in 2·7; being 10 per cent. more fatal than amputations of the *thigh*. This difference depends upon the greater frequency of traumatic amputations of the leg; these, together with the amputations of expediency, being more fatal than those of the thigh. The reason why will be seen as we proceed.

The order of fatality is as follows:

	Fatal.		
1.—Pathological amputations .	. 7·7 per cent,	or 1 in	13
2.—Primary amputations .	. 62·5 „	or 1 in	6
3.—Secondary amputations .	. 66·66 „	or 1 in	5
4.—Amputations of expediency.	. 66·66 „	or 1 in	5

General Conclusions.

From a careful consideration of the whole, together and in detail, the following general conclusions may be drawn:

1. That in amputations of the extremities, taken altogether, 25 per cent. are fatal; 30 per cent. in the lower extremity and 10 in the upper.

2. That amputations, as a whole, are fatal in the following order: Secondary, 50 per cent.; primary, 43 per cent.; amputations of expediency, 30 per cent.; pathological, 12·5 per cent.

3. That in *pathological amputations* of the thigh, 18 per cent. are fatal, or 1 in 5·5; of the leg, 7·7 per cent. are fatal, or 1 in 13. Of the foot and upper extremity success generally follows.

4. That in *amputations of expediency* of the thigh, 31·5 per cent. are fatal, or 1 in 3·16; of the leg, 66·6 per cent. are fatal, or 1 in 1·5. Of the upper extremity fatal cases are exceptional.

5. That in *traumatic amputations* of the *lower* extremity 60 per cent. are fatal; of the *upper* 18 per cent.; and that traumatic amputations of the leg are at least as fatal as those of the thigh.

6. That *secondary amputations* are more fatal than primary.

7. That in amputations of the thigh for chronic disease of the knee-joint, 1 case only out of 7 proves fatal, or about 14·5 per cent. But for acute suppuration a fatal termination is the rule.

8. That in amputations of the *lower* extremity for tumours, 36 per cent. are fatal; after those of the *upper*, recovery may generally be expected.

TABLE D.—*Showing the causes of death after amputation, and number of cases proved per cent. compared with the whole number of fatal cases.*

	Pathological.			Amputations of Expediency.			Primary.					Secondary.				Total.				
	Thigh	Leg.	Total.	Thigh	Leg.	Total.	Thigh.	Leg.	Arm.	Forearm	Total.	Thigh	Leg.	Arm.	Total.	Thigh.	Leg.	Arm.	Forearm.	Total.
Exhaustion from shock of accident, operation, hæmorrhage, or all combined	6, or 33 p. cent.	1, or 33·3	7, or 33·	1, or 16·6	...	1, or 10·	7, or 63·	2, or 15·	...	...	9, or 33·	1, or 33·3	6, or 75·	...	7, or 60·	15, or 40·	9, or 32·	...	...	24, or 33· per cent.
Pyæmia	8, or 44·	1, or 33·3	9, or 43·	3, or 50·	3, or 75·	6· or 60·	3, or 27·	7, or 54·	1, or 50·	1, or 50·	12, or 43·	1, or 33·3	1, or 12·5	1	3, or 25·	15, or 40·	12, or 42·8	2, or 75·	1, or 50·	30, or 42· „
Secondary hæmorrhage	1, or 5·5	1, or 33·3	2, or 9·5	...	...	...	1, or 9·	...	1, or 50·	...	2, or 7·	...	1, or 12·5	...	1, or 8·3	2, or 5·	2, or 7·	1, or 33·3	...	5, or 7· „
Hectic	1, or 5·5	...	1, or 4·7	...	...	...	...	...	...	...	...	1, or 33·3	...	...	1, or 8·3	2, or 5·	...	...	...	2, or 3· „
Cerebral complications	...	...	...	...	...	...	...	1, or 7·	...	1, or 50·	2, or 7·	...	...	...	...	...	1, or 3·5	...	1, or 15·	2, or 3· „
Thoracic complications	1, or 5·5	...	1, or 4·7	...	1, or 25·	1, or 10·	...	2, or 15·	...	...	2, or 7·	...	...	...	...	1, or 2·6	3, or 10·7	...	...	4, or 5·6 „
Abdominal complications	1, or 5·5	...	1, or 4·7	...	...	...	...	...	...	...	...	...	...	...	...	1, or 2·6	...	...	...	1, or 1·4 „
Renal complications	...	...	...	1, or 16·6	...	1, or 10·	...	1, or 7·	...	...	1, or 3·5	...	...	...	...	1, or 2·6	1, or 3·5	...	...	2, or 3· „
Carcinomatous infiltration	...	...	...	1, or 16·6	...	1, or 10·	...	...	...	...	...	...	...	...	...	1, or 2·6	...	...	...	1, or 1·4 „
Traumatic complications	...	...	...	...	...	...	1, or 8·3	2, or 13·3	1, or 33·3	1, or 33·3	5, or 15· *	...	...	...	...	1	2	1	1	5*
Total	18	3	21	6	4	10	12	15	3	3	33	3	8	1	12	39	30	4	3	76

* Excluded in calculation of per centages.

TABLE C.—*Of the different causes of death after amputation, showing their proportions per cent. in the different divisions.*

	Amputations of Thigh.					Amputations of Leg.					Arm.	Forearm.	Total.				
	Pathological.	Expediency.	Primary.	Secondary.	Total.	Pathological.	Expediency.	Primary	Secondary.	Total.	Total.	Total.	Pathological.	Expediency.	Primary.	Secondary.	Total.
Exhaustion from shock of injury, operation, hæmorrhage, or all combined	6·	5·	35·	25·	10·	2·5	...	8·	50·	11·	...	...	4·2	3·3	11·8	29·	8·
Pyæmia	8·	15·7	15·	25·	10·	2·5	50·	32·	8·	14·8	6·4	2·7	5·4	18·	15·8	12·5	10·
Secondary hæmorrhage	1·	...	5·	...	1·4	2·5	...	...	8·	2·4	3·	...	1·4	...	2·6	4·	1·66
Hectic	1·	...	...	25·	1·4	...	...	...	...	...	...	...	·7	...	...	4·	·66
Cerebral complications	...	..	...	...	...	...	...	4·	...	1·2	...	2·7	...	...	2·6	...	·66
Thoracic complications	1·	...	...	...	·7	...	16·	8·	...	3·7	...	...	·7	3·3	2·6	...	1·33
Abdominal complications	1·	...	...	...	·7	...	...	...	...	...	...	...	·7	...	...	...	·33
Renal complications	...	5·	...	...	·7	...	...	4·	...	1·2	...	...	...	3·3	1·3	...	·66
Carcinomatous infiltration	...	5·	...	...	·7	...	...	...	...	...	...	...	...	3·3	...	...	·33
Traumatic complications	...	...	5·	...	·7	...	...	8·	...	2·4	3·	2·7	...	...	6·5	...	1·66
Total fatality	18·	31·5	60·	75·	27·2	7·7	66·6	62·5	66·6	37·	12·9	8·3	12·5	30·	43·	50·	25·3

Analysis of Tables B and C.

Causes of Death.

Pathological amputations.—We will now proceed to the more immediate subject of the causes of death after amputations; and reviewing the 21 cases of death after pathological amputations of the thigh and leg, we find that, analysing the 18 cases of fatal amputations of the thigh, 8, or 44 per cent., died from pyæmia upon the fifth, seventh, eighth, eighth, ninth, eleventh, twelfth, and forty-seventh days; 6, or 33 per cent., from exhaustion upon the fourth, eighth, eighth, eighth, twelfth, and nineteenth days respectively; 1 had secondary hæmorrhage upon the twelfth day, and died on the twenty-second, with diseased kidneys; 1 sank from diarrhœa upon the twenty-first day; 1 from diarrhœa and phthisis upon the thirtieth; 1 from hectic upon the twenty-second day.

Four of the 8 cases which died from pyæmia, were amputated for acute suppuration of the knee-joint, produced in 2 by the discharge of an abscess into the joint; in the remaining 4 amputation was performed for chronic disease of the joint. Out of the remaining 10 cases of death, 9 were amputations for chronic disease of the knee, and 1 for gangrene of the leg after ligature of the femoral artery for ruptured aneurism.

From the above, it would appear that amputation for acute suppuration of a joint is more frequently followed by pyæmia than amputations for chronic disease.

Of the 3 fatal cases of amputations of the leg, 1 died from secondary hæmorrhage upon the fifth day; 1 from exhaustion upon the twenty-fifth day; 1 from pyæmia upon the ninth day.

Reviewing these cases as a whole, and comparing Tables B and C together, we find that, out of the 21 examples of *fatal* pathological amputation of the lower extremity—

	Of fatal cases.	Of whole number.
Pyæmia was the cause of death in	43· per cent.,	and 6·4 per cent.
Exhaustion „ „	33· „	5· „
Secondary hæmorrhage „	9· „	1·4 „
Hectic, Abdominal and Thoracic complications „ each	4·5 „	·7 „

Amputations of expediency.—Under this heading we have but 10 deaths, the operations being less frequent than in the former division. 6 of these are of the thigh, and 4 of the leg.

Of the fatal thigh cases, 1 sank exhausted nine hours after the operation; 1 from diseased kidneys; 1 from infiltration of carcinoma; 3 from pyæmia, 1 upon the third day, and 2 from the secondary abscesses in the sixth and twelfth weeks. In 5 of these cases the limb was removed for tumours of the bone, and in 1 for elephantiasis.

Of the 4 fatal leg cases, 3 died from pyæmia upon the fifth, thirteenth, and thirteenth days respectively; 1 from pneumonia and bed-sore upon the thirtieth day. Two were cases of talipes, 1 of elephantiasis, 1 of tumour, and 1 associated with diseased bone.

In both the cases of elephantiasis, death followed from pyæmia, upon the third and fifth days.

Taking these amputations of expediency together, 30 per cent. were fatal; 18 per cent. from pyæmia, 3 from exhaustion, and the remaining 9 from visceral complications.

But as, by the table, it is seen that this operation, when performed upon the upper extremity, is generally followed by success, the average of the mortality of the lower becomes 40 per cent. The causes of death, and their proportion both to the fatal cases and to the whole number amputated, will be seen by the following table:

	Per centage of			Per centage of		
	Fatal cases.		Whole number.	Fatal cases.		Whole number.
Exhaustion proves fatal in	Thigh 16·6	..	5·	Leg —	..	—
Pyæmia „ „	50·	..	15·7	„ 75·	..	50
Visceral complications „	33·	..	10·	„ 25·	..	16·

Conclusion. — Amputations of expediency of the leg are twice as fatal as those of the thigh; pyæmia, the most fatal cause of death, being 50 per cent. more fatal in amputations of the leg than of the thigh, and destroying half the cases of the former operated upon. Exhaustion and visceral complications caused death in the remainder.

Primary amputations.—Amongst these cases are 33 fatal examples; 12 being of the thigh, 15 of the leg, 3 of the arm, and 3 of the forearm.

Analysing the 12 cases of the thigh, 7 died from *exhaustion*, whether produced by the shock of the accident, of the operation, or from hæmorrhage, or all combined, upon the third or fourth days; 3 died from pyæmia upon the twenty-sixth, twenty-seventh, and twenty-eighth days, symptoms appearing upon the seventh, sixteenth, and twentieth days; 1 died from secondary hæmorrhage, which took place upon the third day, and proved fatal upon the eighth day; 1 from traumatic complications, as fractured ribs and ruptured spleen.

Amongst the 15 fatal cases of amputations of the leg, 2 sank from *exhaustion* upon the third day; 7, or 46 per cent. of fatal cases, from *pyæmia*—all dying about the twenty-fifth day, ten days after the appearance of the symptoms; 2 died from *pneumonic* complications—one from acute bronchitis on the twentieth day, and the second from pneumonia on the seventeenth day; 2 died from severe traumatic complications upon the third and eighth days; 1 from diseased kidneys upon the ninth day; 1 from delirium tremens upon the fifth day.

Analysing the fatal cases of amputation of the arm, 1 died from the traumatic complication of compound fractured thigh upon the fourth day; 1 did well till the thirteenth day, when sloughing appeared, followed upon the twenty-first by secondary hæmorrhage, and death on the twenty-second day; 1 died from pyæmia upon the thirtieth day, fourteen days after the appearance of the symptoms.

Proceeding to the fatal amputations of the forearm, 1 died from pyæmia upon the twenty-third day, symptoms appearing upon the fourteenth day; 1 from ruptured kidney upon the sixteenth day; 1 upon the fourth day from delirium tremens, associated with old arachnitis, the hemispheres being adherent at the median line.

Analysing the whole number of fatal primary amputations, amounting to 33 cases, 12, or 43 per cent., died from *pyæmia;* 9, or 32 per cent., from exhaustion; 5 died from visceral complications; 5 from traumatic complications; 2 from secondary hæmorrhage.

Taking out of our calculation the 5 cases which died from some traumatic complications, there remain 28 fatal examples, 43 per cent. of which proved fatal from *pyæmia;* 32 per cent. from *exhaustion;* 25 per cent. from visceral complications or secondary hæmorrhage.

On comparing these causes of death with the whole number of amputations, 16 per cent. died from pyæmia, 12 per cent. from exhaustion.

Primary amputations of the thigh prove for the most part fatal from exhaustion, produced by the accident, the operation, or both combined; two. thirds of the cases dying from such a cause. The remaining third is caused by pyæmia.

In primary amputations of the leg, exhaustion has but a small influence in causing a fatal termination, but *pyæmia* becomes considerably more fatal, about half the fatal cases dying from such a cause. Visceral complications destroy the remainder.

In the primary amputations of the upper extremities, pyæmia is the chief cause of death, together with some accidental visceral complications.

Taking primary amputations of the *thigh* alone, 60 per cent. are fatal; *exhaustion* proving the fatal cause in 35 per cent., *pyæmia* in 15 per cent., and secondary hæmorrhage and traumatic complications in 5 per cent. each.

Taking primary amputations of the *leg,* 62 per cent. are fatal; *pyæmia* being the cause of death in 32 per cent.,

exhaustion, traumatic, thoracic, and visceral complications, in 8 per cent. each.

Comparing the two together, amputations of the thigh and leg are equally fatal (about 60 per cent.); but *pyæmia* is twice as fatal in amputations of the leg as of the thigh, and in these latter *exhaustion* is the chief cause of death.

Secondary amputations.—The 12 fatal examples include 3 of the thigh, 8 of the leg, and 1 of the arm.

Thigh.—One sank upon the sixth day from exhaustion; 1 upon the ninetieth from hectic and suppuration; 1 upon the twelfth from pyæmia, symptoms appearing upon the tenth.

Leg.—Four sank upon the fifth, fifth, ninth, and tenth days, in 1 diseased kidneys being found; 2 upon the eighth day, with gangrene of stump; 1 upon the second day, after secondary hæmorrhage; 1 upon the twentieth day, from *phlebitis*.

Arm.—One died upon the ninth day from *pyæmia*, symptoms appearing upon the fifth.

Analysing the whole number of cases, 7, or 60 per cent., sank after the operation from *exhaustion;* 3, or 25 per cent., from *pyæmia;* 1 died from secondary hæmorrhage; 1 from hectic.

Taking secondary amputations as a whole, a very different result to that obtained by an analysis of the primary may be educed from the consideration of the preceding facts.

Out of the 12 fatal cases, about 60 per cent. die from exhaustion; 25 per cent. from pyæmia; 15 per cent. from hectic and secondary hæmorrhage.

Exhaustion is here the chief cause of death, and not pyæmia, which sinks in proportion.

In secondary amputations of the *thigh*, 75 per cent. are fatal; exhaustion, pyæmia, and hectic being equally fatal causes.

In secondary amputations of the leg, 66 per cent. are fatal; 50 per cent., or half those operated upon, dying from

exhaustion; pyæmia and secondary hæmorrhage being fatal in only 8 per cent. each.

In *traumatic amputations of the upper extremity*, about 20 per cent. are fatal; pyæmia is the chief cause of death, one half dying from this cause; traumatic or some other accidental visceral complications prove fatal in the remainder.

Analysing the tables of the causes of death after amputation as a whole, and taking out of our calculations the cases which die from other injuries received at the time, we may fairly conclude that, amongst the 71 fatal cases—

42 per cent. die from pyæmia; 33 per cent. from exhaustion; 20 per cent. from visceral complications; 7 per cent. from secondary hæmorrhage. Hectic proves but a small cause of death, occurring in but 2 cases out of the 71, or not 3 per cent.

40 per cent. of all fatal amputations of the *thigh* die from *exhaustion*, and the same average from *pyæmia*.

32 per cent. of fatal amputations of the *leg* sink from *exhaustion*, and 42 per cent. from *pyæmia*. Secondary hæmorrhage, hectic, and visceral complications divide between them the other 30 per cent.

Comparing the same causes of death with the whole number of amputations, it will be seen that 25 per cent. are fatal, including 10 per cent. from *pyæmia*, 8 per cent. from *exhaustion*, 1·66 per cent. from secondary hæmorrhage, 1·66 per cent. from traumatic complications, and the remaining 5 per cent. from hectic and visceral complications.

Comparing *pyæmia* and *exhaustion* as causes of death after amputation generally, the following table will be of value, as showing the proportion they bear to one another:

In all *fatal* cases of

	Exhaustion is the cause in	Pyæmia is the cause in
Amputation of the extremities	.. 33· per cent.	.. 42· per cent.
Pathological amputation	.. 33· „	.. 43· „
Amputations of expediency	.. 10· „	.. 60· „
Primary amputation ..	.. 32· „	.. 43· „
Secondary „ ..	.. 60· „	.. 25· „

It will be observed, that in secondary amputations exhaustion is the most fatal cause, and in amputations of expediency the least fatal; and that in pathological and primary amputations the general average is maintained.

On the other hand, it will be seen that, regarding pyæmia as a cause of death, the above is exactly reversed; pyæmia being most fatal in the amputations of expediency, and the least so in secondary amputations. In pathological and primary amputations the general average is the same.

Comparing these fatal causes with the whole number of amputations, it will be seen in Table C that exhaustion is the cause of death in 8 per cent., and pyæmia in 10 per cent. That *exhaustion* is most fatal in secondary, and the least so in amputations of expediency, and that it destroys 12 per cent. of primary, and 4 per cent. of pathological amputations. *Pyæmia*, on the other hand, is most fatal in amputations of expediency, and the least so in secondary amputations.

Comparing the same causes, viz., pyæmia and exhaustion, as bearing upon the fatality of amputations of the *thigh* and *leg*, some interesting conclusions may be gathered from the following table:

	Pyæmia as a fatal cause.		Exhaustion as a fatal cause.	
	Thigh. Per cent.	*Leg.* Per cent.	*Thigh.* Per cent.	*Leg.* Per cent.
In fatal pathological amputations	44· ..	33· ..	33· ..	33·
„ amputations of expediency	50· ..	75· ..	17· ..	—
„ primary amputations	27· ..	54· ..	58· ..	13·
„ secondary „	33· ..	12· ..	33· ..	75·
„ amputations generally	40· ..	42· ..	40· ..	32·

In pathological amputations of the thigh, *pyæmia* appears

to be a more frequent cause of death than in the same operation upon the leg; in amputations of expediency, it would appear that pyæmia is 25 per cent. a more frequent cause of death when applied to the leg than to the thigh; in primary amputations the difference is still more marked, for pyæmia is a cause of death in such operations upon the leg in the proportion of 54 per cent., and in the thigh but 27 per cent., or exactly twice as fatal.

In secondary amputations the reverse must be observed, for in the thigh, pyæmia is a cause in 33 per cent., in the leg, but 12 per cent.

From Table C similar conclusions may be drawn.

Touching the subject of *exhaustion* as a cause of death in cases of amputation of the thigh and leg, it bears exactly an opposite proportion to pyæmia; for when the numbers are high under exhaustion, they are low under pyæmia.

In *pathological amputations* they bear an equal average. In *amputations of expediency*, in which pyæmia is so fatal when applied to the leg, exhaustion is an unknown cause; and in the thigh it is the cause of death in but 17 per cent. of the fatal cases. In *primary amputations* of the thigh, exhaustion is the cause of death in 58 per cent.; of the leg in 13 per cent. only; being the reverse of pyæmia, which is 100 per cent. more fatal in amputations of the leg than of the thigh. In *secondary amputations*, again, exhaustion proves more fatal in the leg than in the thigh, in the proportion of 75 to 33 per cent.

Period of death from pyæmia after operation.—Amongst the 33 fatal examples of pyæmia, there are 15 cases following pathological amputations and amputations of expediency of the lower extremity; 12 of these died within fourteen days, the remaining 3 sinking from secondary abscesses in the second or third month. The symptoms made their appearance within a few days of the operation.

Among traumatic amputations there are 12 cases, 1 only of these dying upon the twelfth day, the remaining

11 cases sinking upon the twenty-fifth or twenty-seventh days.

In the upper extremity but 3 cases occurred, death taking place upon the ninth, twenty-third, and thirtieth days. The symptoms, as a rule, appeared from seven to fourteen days after the operation.

The difference in the duration of the disease called pyæmia, in the two classes of cases just mentioned, is worthy of observation; and although I am unable to explain why, after traumatic amputations, the patients sinking from pyæmia should live twice as long as in pathological amputations and those of expediency, the fact remains, and additional observations are required to solve the difficulty.

General Conclusions upon the Causes of Death in Amputations.

1. That 25 per cent. are fatal; 30 per cent. of the lower extremity, 10 per cent. of the upper.

2. That *pyæmia* is the cause of death in 42 per cent. of the fatal cases, and in 10 per cent. of the whole number amputated.

3. That exhaustion is the cause of death in 33 per cent. of the fatal cases, and in 8 per cent. of the whole number amputated.

4. That the following causes of death are fatal in the annexed proportions:

	Of fatal cases.	Of whole number.
Secondary hæmorrhage	7· per cent.,	or 1·66 per cent.
Thoracic complications	5·6 „	„ 1·33 „
Cerebral „	3· „	„ ·66 „
Abdominal „	1·4 „	„ ·33 „
Renal „	3· „	„ ·66 „
Hectic „	3· „	„ ·66 „
Traumatic „	7· „	„ 1·66 „

Pathological Amputations.

1. That pathological are by far the most successful amputations, only 12·5 per cent. proving fatal; such amputations of the upper extremity are generally followed by success, of the lower extremity 15 per cent. terminate fatally.

2. That *pyæmia* is the chief cause of death, proving fatal in 43 per cent. of the fatal cases, and in 5·4 per cent. of all pathological amputations; and when fatal, as a rule, it causes death within fourteen days of the operation.

3. That *exhaustion*, either from the shock of the accident or of the operation, from hæmorrhage, or all these causes combined, is the cause of death in 33 per cent. of the fatal cases, or 4 per cent. of all amputations.

4. That *secondary hæmorrhage* is the fatal cause in only 9 per cent. of the fatal cases, and in 1·4 per cent. of all amputations.

5. That hectic, abdominal and thoracic complications, act equally as causes of death in 13 per cent. of the fatal cases, and in 2 per cent. of all amputations.

Amputations of Expediency.

1. That 30 per cent. are fatal; but as amputations of the upper extremity are, as a rule, successful, the per centage of this operation upon the lower is much increased, 40 per cent. proving fatal.

2. That *pyæmia* is the chief cause of death, proving fatal in 60 per cent. of the fatal cases, and in 18 per cent. of all such amputations; and when fatal, as a rule, death takes place within 14 days of the operation.

3. That death from *exhaustion* occurs in but 10 per cent. of the fatal cases; and that some thoracic or renal complications, or carcinomatous infiltration, are fatal causes in the same proportion.

Primary Amputations.

1. That 43 per cent. are fatal; 60 per cent. of the lower extremity, and 30 per cent. of the upper.

2. That primary amputations are more successful than secondary.

3. That *pyæmia* is the cause of death in 43 per cent. of the fatal cases, and in 16 per cent. of the whole number; and that, when fatal, the symptoms appear, as a rule, between the seventh and fourteenth days after the operation, and cause death in the third or fourth week, and not during the first two weeks, as in pathological amputations and those of expediency.

4. That *exhaustion* is the cause of death in 32 per cent. of the fatal cases, and in 12 per cent. of the whole number.

5. That *traumatic complications* prove fatal in 15 per cent. of the fatal cases; and secondary hæmorrhage, cerebral or thoracic complications, in about 7 per cent. each; renal disease proving a cause of death in 3·5 per cent.

Secondary Amputations.

1. That 50 per cent. are fatal; 68 per cent. of the lower extremity, and 12·5 per cent. of the upper.

2. That secondary amputations are more fatal than primary by about 8 per cent.

3. That *exhaustion* is the chief cause of death, proving the cause in 60 per cent. of the fatal cases.

4. That *pyæmia* is the cause in 25 per cent. of the fatal cases; secondary hæmorrhage and hectic in the remaining 15 per cent.

CONCLUSIONS UPON PYÆMIA AS A CAUSE OF DEATH.

1. That it is the cause of death in 42 per cent. of all fatal cases of amputation, and in 10 per cent. of all amputations.

2. That it is the cause of death in the different forms of amputation in the following order: in 70 per cent. of all fatal amputations of expediency; in 43 per cent. of all fatal primary amputations; in 43 per cent. of all fatal pathological amputations; in 25 per cent. of all fatal secondary amputations; and that in amputations of expediency it is the most frequent cause, in secondary amputations the least.

3. That in amputations for acute suppuration of the knee-joint, whether the result of an abscess discharging into the joint or otherwise, pyæmia is a more frequent cause of death than in amputations for chronic disease.

4. That it is the general cause of death in amputations for talipes, elephantiasis, and tumours.

5. That in primary amputations, and in amputations of expediency of the leg, it is a more frequent cause of death than in the same operations upon the thigh.

6. That upon the whole, pyæmia appears to be a more frequent cause of death in amputations through limbs, the tissues of which are in a normal condition, and where a large surface of healthy bone is exposed.

7. That in pathological amputations, and in amputations of expediency, pyæmia, as a rule, proves fatal within fourteen days; but after traumatic amputations, the period of death is about the twenty-fifth or twenty-sixth day.

General Conclusions *upon Amputations of the Thigh.*

1. That 27 per cent. are fatal: of pathological amputations 18 per cent.; amputations of expediency 31 per cent.; primary amputations 60 per cent.; secondary amputations 75 per cent.

2. That in amputations of the thigh for chronic disease of the knee-joint, about 15 per cent. are fatal, or 1 case in 7.

3. That amputations of the thigh for acute suppuration

in the joint are generally fatal, and that pyæmia is the chief cause of death in these cases.

4. That exhaustion and pyæmia are causes of death in equal proportions, or in about 40 per cent. of the fatal cases, and in 10 per cent. of all amputations of the thigh.

5. That *exhaustion* is most fatal in primary amputations, and the least so in amputations of expediency.

6. That *pyæmia* is most fatal in amputations of expediency, and the least so in primary.

7. That primary amputations for the most part are fatal from exhaustion, 35 per cent. of the cases sinking from this cause; 15 per cent. from pyæmia; and from secondary hæmorrhage and traumatic complications 5 per cent. each.

8. That exhaustion, pyæmia, and hectic are equally fatal causes in secondary amputations, proving fatal in 25 per cent. each.

Amputations of the Leg.

1. That 37 per cent. are fatal: of pathological amputations 7·7 per cent.; amputations of expediency 66·6 per cent.; primary amputations 62·5 per cent.; secondary amputations 66·6 per cent.

2. That amputations of the leg are 10 per cent. more fatal than of the thigh; the amputations of expediency and traumatic amputations being more fatal, and the latter more frequent.

3. That amputations of expediency of the leg are generally fatal, being twice as fatal as those of the thigh. That pyæmia is the chief cause of death in 75 per cent. of the fatal cases, and in 50 per cent. of all such amputations.

4. That in primary amputations, pyæmia is the cause of death in half the fatal cases, or in 32 per cent. of all such operations; exhaustion and visceral complications about 8 per cent. each.

5. That, comparing primary amputations of the thigh and leg together, they are equally fatal; but that pyæmia is twice as fatal in amputations of the leg as in amputations of the thigh.

6. That half the cases of secondary amputation die from exhaustion; pyæmia and secondary hæmorrhage being fatal in 8 per cent. each.

7. That taking all amputations of the leg together, 42 per cent. of the fatal cases die from pyæmia, and 32 per cent. from exhaustion.

Amputations of the Upper Extremity.

1. That 10 per cent. are fatal.

2. That pathological amputations, and those of expediency are, as a rule, successful.

3. That about 20 per cent. of traumatic amputations are fatal; 22 per cent. of the arm, and 16 per cent. of forearm.

4. That one third of these fatal cases die from pyæmia, one third from some traumatic complication, and the remaining third from secondary hæmorrhage or visceral disease.

ON

SOME OF THE CYCLICAL CHANGES

IN THE

HUMAN SYSTEM

CONNECTED WITH SEASON.

BY

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I have occupied the greater part of the last four years in an endeavour to determine the cyclical changes in the chief function of the body, viz., the respiration, and have, to some extent, succeeded in my object. Two papers have been published in the 'Transactions of the Royal Medical and Chirurgical Society' for 1856, in which I endeavoured to show the cyclical changes of the twenty-four hours, hour by hour, so far as relates to the rate of pulsation and respiration in health and in phthisis; and a paper showing the variations in certain phenomena during the period of a month in the summer season, was published in the 'British and Foreign Medico-Chirurgical Review' in April of the same year. Since their publication I have ascertained the hourly changes in the quantity of air inspired and the

carbonic acid expired, with other phenomena of respiration, and have shown that the changes in the two latter subjects of inquiry are very similar to those of the rate of these functions, and that the progression of the day is as follows. Commencing in the depth of the night, at 1 to 3 a.m., when the function is at the lowest point of the twenty-four hours, there is a gradual and slow increase up to the usual period of rising, when it is at the lowest point of the daylight day—a point representing 75 per cent. of the whole average action of the twenty-four hours. After each meal, during the day, there is a rapid and progressive increase during about two and a half hours, to a maximum of about 35 per cent. of the quantity before breakfast, and then a more rapid decline, until the minimum of the daylight day is again obtained shortly before each meal. In the evening, however, there is seldom any increase after the supper; but whatever may be the existing circumstances, the changes diminish progressively, and at length fall very rapidly, until the minimum of the twenty-fours (viz., half of the maximum) is again attained. Thus, there is an unintermitting cycle of changes through the day and night coexistent with various physical phenomena, but not altogether explicable by them, in which the excess of action of the day alternates with an equal defect through the night.

I have now extended the same inquiry to the changes of the system in health from day to day, and month to month, through the cycle of the seasons, and have found, that however great and important the changes of the day may be in reference to the maintenance of the health and the fulfilment of the duties of the body, those of season are even more so, since they enable us to obtain a glimpse at some of the fundamental laws of the system. In presenting to the Society a short account of the results obtained, I purpose to avoid, as far as possible, extended physiological detail, and to limit myself to a short statement of the results and the deductions which flow from them.

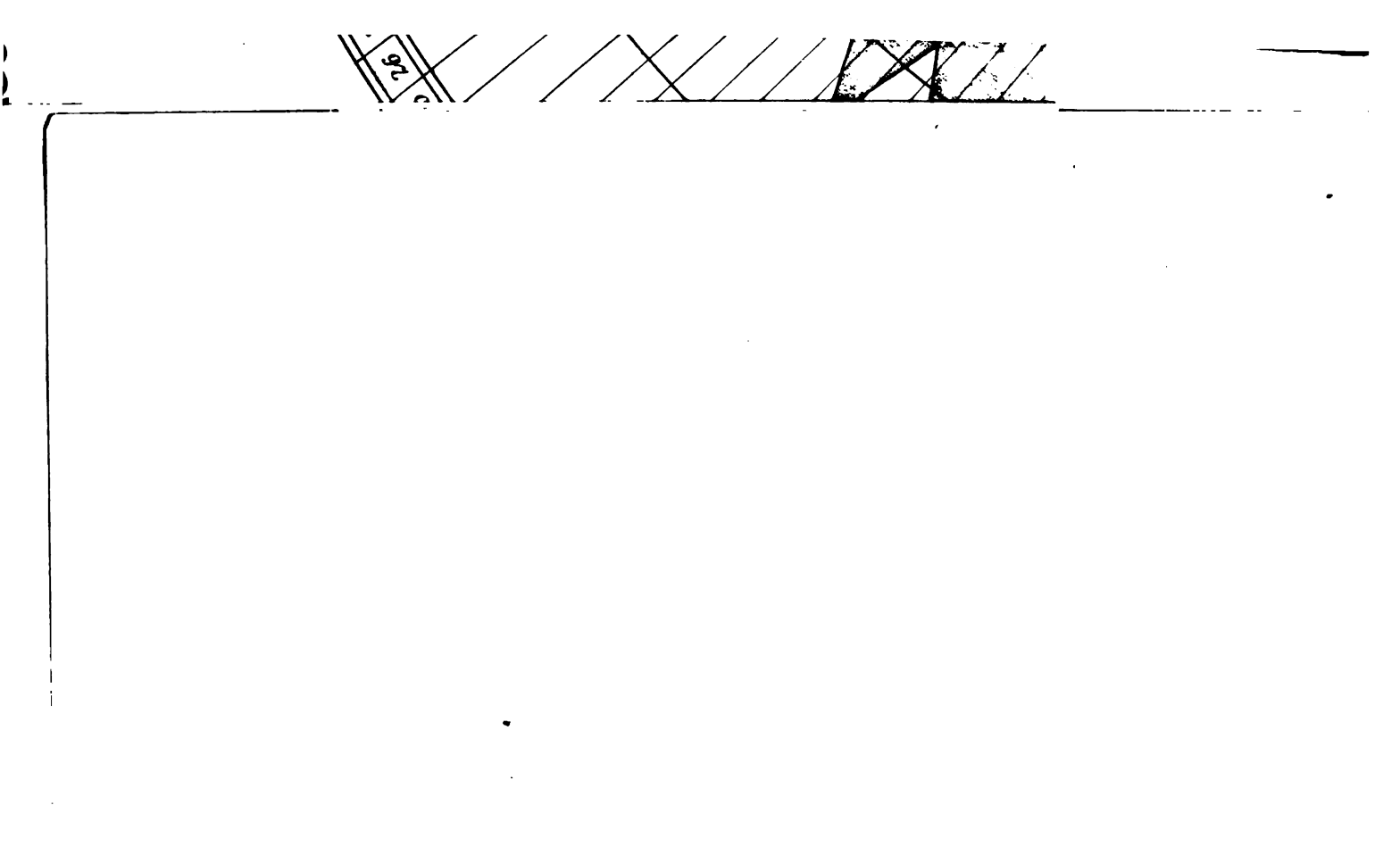
Historical sketch.—Although the inquiry in the serial

and extended form now mentioned is new, we have obtained from observers in physiology and practical medicine a large amount of knowledge which bears upon this subject. Thus, all physiological observers have noticed that the respiratory changes were never the same on any succeeding days. Vierordt has proved that they vary inversely, as the temperature and height of the barometer. Lavoisier and Liebig have both asserted that this function is less active in summer than in winter, partly because with heat the volume of air is increased, and thereby less oxygen remains in a given volume (omitting, however, all reference to the whole amount of air which is inspired), partly because the necessity for it to maintain the animal heat is lessened, and partly because it had been observed that the quantity of food taken was diminished. In my former inquiries I showed that the rate of respiration was lessened, and that of pulsation increased, with increasing temperature. It is believed, that in hot climates the respiratory function is lessened, whilst the function of the liver is increased in activity, and the diseases of those climates refer to the latter rather than to the former organ. Languor and relaxation are familiarly believed to prevail more in hot seasons and climates than in cold; and change of the weather towards the opposite, and therefore in all directions during the cycle of the seasons, is matter of common congratulation. Diseases vary in intensity, type, and frequency with season and climate, and even the action of remedies is believed to vary under those circumstances. Moreover, certain diseases are almost limited to certain seasons, and the various seasons as they complete their cycle bring with them causes of special liability to the production of disease. Certain animals gain weight in summer and lose it in winter, and the same fact has very recently been proved to occur in the prisoners at the convict gaol at Wakefield, where, for a series of ten years (as has been shown by the surgeon, Mr. Milner), they gain weight from May to September inclusive, and lose weight during the other months of the year—the diet and labour being the same throughout the year, and

the periods of change being abrupt in April and October. Hence, without entering at length into the literature of this subject, it may be affirmed that much knowledge has been gained, and that what is lacking is chiefly in extent and also in demonstration on subjects on which we have as yet only opinions and inferences, and the determination of the laws and conditions upon which these cyclical changes depend.

Description of the method and apparatus.—The inquiry which I shall now proceed to describe has been made upon myself, æt. 39, and a gentleman in perfect health, æt. 48—the one of us bearing great heat well, and the other suffering much from it: it was commenced on March 31st, 1858, and has been continued on about one hundred and fifty occasions to the present time. Each experiment has been made under precisely similar and normal circumstances, viz., between 7 and 8 a.m., before breakfast, in the sitting posture, and in the absence of all bodily exertion and mental excitement. The experiments may, therefore, be compared with each other; and since the disturbing influence of disease, food, and exertion was avoided, the variations in the state of the system must be chiefly due to season.

The apparatus employed (See Diagram in 'Phil. Trans.,' 1859) was a spirometer, already described to the Society, to measure the inspired air, and an analytical apparatus, whereby all the vapour and the carbonic acid could be removed from the expired air during the act of expiration, and for any period, however long or short. The latter consisted of a gutta-percha box, divided into chambers, and each chamber into cells, in such a manner that each column of one cubic inch of air was made to traverse, in a given direction, a surface of seven hundred superficial inches. The floor of each chamber was covered with a solution of caustic potass of sp. gr. 1·27, in order to absorb the carbonic acid; and as the expired air was first dried before it entered this box, the vapour which it carried away at its exit was arrested



a second drying apparatus. The addition to the weight the box and the second drying apparatus gave the antity of carbonic acid abstracted from the expired air. he balances employed were made by Oertling, and igh to the $\frac{1}{100}$th of a grain, with 7 lbs. in each pan. here was no impediment to free expiration.

Results of the inquiry—The results which have been tained are very decided, and the progression in the anges of the system are so uniform, and pursue a course consonant with common and professional observation, at I think no valid objection can be taken to them. It true that they have been derived from two persons only—at these two belong to the two classes into which mankind may be divided in reference to the degree of tolerance heat; and, although variations in period and degree may found in others, it is very probable that the general progression which our returns show would still be applicable others. It is this general progression in the cycle of anges, rather than the amount of change, which I think important to demonstrate; and after having made some ousands of experiments more than any other observer as recorded, I have arrived at the opinion expressed by at laborious inquirer, Vierordt, that we can only obtain a rge number of trustworthy observations from experiments ng continued upon a few persons.

The general expression of the results obtained (*see* iagram) is that, as the season advanced from spring to ummer and autumn, all the respiratory phenomena were essened, viz., the volume and much more the weight of air spired, the amount of vapour and carbonic acid exhaled, he rate of respiration, the cooling of the body, and the uscular and vesicular actions of respiration. The highest tate of the functions was in spring; the period of decline was the beginning of summer; the lowest state was towards he end of summer and in the beginning of autumn; and he period of increase was the beginning of winter. The ighest months were April and May; the lowest were part

of July, August, and part of September. The difference at these various periods was large and decided. Thus the extreme difference in myself between the highest and the lowest periods, when taken on the average of a month, was 30 per cent. of the larger quantity, in the quantity of air inspired, 32 per cent. in the rate of respiration, and 17 per cent. in the quantity of carbonic acid exhaled. In Mr. Moul, to whom I am indebted for having become the other subject for experiment, the diminution so early as the middle of June, when his experiments terminated, was 27 per cent. in both the carbonic acid expired and the air inspired, and 28 per cent. in the rate of respiration. He exhibited more quickly and in a greater degree than myself the effects of season at this comparatively early period, in accordance with an observation which I published in connexion with former inquiries, viz., that those who suffer much from heat have an excess of all the seasonal changes. Hence season acts with varying power and rapidity upon different persons.

These various results are delineated upon the Diagram accompanying this paper. This shows, in a circle, the number of grains of carbonic acid evolved, and the degree of temperature of the external air on many days in each month of the year. The outer lines show the carbonic acid evolved by myself and Mr. Moul, and the inner line the temperature.

There are, therefore, two seasons of the year in each of which the respiratory phenomena are comparatively uniform for a considerable period, and two others which are truly seasons of change. The former are, in general terms, the winter and summer seasons, and the latter the spring and autumn seasons; and as it will be convenient to give to each of them a short designation, I purpose to speak of them as the maximum, decreasing, minimum, and increasing periods, seasons, and states of system. In order, however, to speak of the subject more accurately, it is necessary to refer to shorter periods than three months, and to arrange the months as follows:

Fixed.	Variable.
Maximum—Jan., Feb., March, April, May.	Decreasing—June.
Minimum—July, Aug., Sept. (part of).	Increasing—Oct., Nov., Dec.

In some persons, and probably in some seasons also, May and September must be included in the variable, and December in the fixed.

Thus there is a cycle of changes proceeding from month to month, having definite periods of maximum and minimum intensity, and of increase and decrease, but doubtless varying within limits in different persons, and under the varying degrees of power of the causes to which they are due. These causes are certainly, in great part, the external influences which constitute the seasons; but it is quite possible that there is a law implanted in our nature which in other part governs these changes, and which we shall not be able to refer to the effects of external agencies. Certain it is that the meteorological influences which are associated with the season are insufficient to fully explain the changes which have been described, as the following statement will prove.

Relation to physical phenomena.—The relation of these changes to *temperature* is not precisely that which has been hitherto received. With sudden accession of heat there was a sudden diminution of the respiratory changes, but in such a manner that, with increasing temperature, the respiration fell not in the same, but in an *increasing ratio*. With subsequent diminution of temperature the respiration suddenly rose. Hence, in both directions, whether tending to health or disease, the change was disproportionately powerful, in accordance with common observation. This was the most marked in the first great and temporary increase of temperature after the long-continued cold of the winter; and in subsequent accessions during the period of increasing temperature, whilst the direction of the effect was always the same, the degree of it diminished. A medium temperature, such as that at which we endeavour

to keep our rooms throughout the year (which is, therefore, the most agreeable to our system in this climate), and such as is found in the open air at three of the four seasons of the year, viz., from 55° to 60°, is accompanied by all the conditions of the respiration, and which varied in myself from 9·13 grs. to 6·76 grs. of carb. acid per minute. Hence, in that degree, temperature and respiration do not maintain any fixed relation. Above 60° temperature had a more decided influence; but, whatever was the degree of heat, as the season advanced towards August and the beginning of September, the respiratory changes decreased month by month. Thus it is clear that numbers of degrees of temperature bear only a very indefinite relation to the respiratory changes. It is also the fact that the system may become accustomed to the continued influence of a given temperature, and then will exhibit the effect of temperature chiefly at the first sudden change.

The effect of barometric pressure was very similar to that of temperature. The barometer was the lowest at the period of maximum respiration; but a medium height, as between 29 and 30 inches, was accompanied by every variation in the respiratory changes.

The degree of saturation of the atmosphere with vapour had but a very general correspondence with the results obtained from the respiration, but with low temperature and high respiration the degree of saturation was greater, and the contrary with high temperature and low respiration. As, however, the total quantity of vapour contained in the air inspired under the latter circumstances was much greater than with the low temperature, the quantity which could be removed from the lungs by expiration was lessened; and this diminution was further increased by the greatly lessened rate of respiration and the quantity of air inspired.

A review of these three agencies cannot fail to show that the influence of season is much more than can be ascribed to them. There are two other agents which are known to be powerful, but with which we are too imper-

fectly acquainted to enable me at present to apply them to this subject, viz., light and magnetism. But without hazarding any conjectures as to the precise agencies to which the influence of season should be attributed, there can be no doubt of the truth of the fact which I desire to prove to the Society, viz., that, as the season advances towards autumn, all the respiratory phenomena are lessened, and that they do not reassume an upward tendency until long after the maximum period of temperature has passed over.

Relation to the production and cure of disease.—Such are the facts obtained by me in reference to the respiratory changes; and before I apply them to the production of disease I would meet two objections which would doubtless be urged against their application, viz., that the determination of the amount of carbon emitted by the lungs does not include all the carbon which passes out of the body; and, whatever may be the variations in reference to the carbon, it does not follow that there are similar variations in the plastic changes of the system.

In reference to the first, I reply, that I have, in a certain degree, determined the amount of carbon emitted by the skin (the organ which is loosely believed to act to some extent as vicarious of the lungs), and I find that in July, viz., at a period approaching the minimum period of the respiration, the amount of carbonic acid exhaled per hour by my skin did not exceed six grains—a quantity so disproportionately small when compared with the 500 to 600 grains exhaled per hour by the lungs, that, for practical purposes, in health, the skin cannot be regarded as vicarious of the lungs in reference to chemical changes.

This experiment was made by inclosing the whole body, except the head, in a caoutchouc dress, and passing a known volume of air through the bag so as to maintain the skin in its normal state, the whole air being made subsequently to traverse the analytical apparatus. In reference to the other excretions of carbon and also of nitrogen, I quote the

results obtained by Barrall in 1848 and 1849. He ascertained during five days in winter, and again in summer, the exact composition and quantity of his food and the composition of his excretions during the period of inquiry, and he found that there was great difference in all the results at the two periods of the year, and in such a manner that the total amount of both carbon and nitrogen in the excretions was lessened in summer, and lessened somewhat proportionately both to the food and to the carbon expired in respiration. Hence, for my purpose, when I prove that the respiratory changes are varied with season, I have a right to say that the vital actions in the system change in like manner. In Barrall's experiments the diminution in the carbon excreted per day, exclusive of that by the lungs and skin, was from 470 grains to 364 grains; and of nitrogen from 210 grains to 182 grains. The variation in the amount of food taken also supports the above assertion.

Thus, the variations in the system, in the summer, may be tabulated as follows:

1. Lessened respiratory and other chemical changes.
2. Usually increased rate of pulsation with increase of blood at the superficies, as shown by the tightness of certain articles of clothing and the colour of the skin; and, as a consequence, a diminished quantity remaining at the centres.
3. Increase in the function of the skin, viz., the transpiration of vapour, and increased sensibility to cold.
4. Lessened difficulty in maintaining a sufficient degree of animal heat, but greater difficulty in preventing excess.
5. Lessened appetite and lessened quantity of food, as proved by Barrall's experiments, in whom the diminution was 28 per cent. in the food taken, and 23 per cent. in the carbon excreted, apart from the lungs and skin.
6. There must be changes in the state of the blood, in reference to the degree of oxidation, the quantity of fibrin,

and the degree of alkalinity; but these have not been sufficiently inquired into.

7. It is probable that there is increased action of the liver.

Such and so great being the variations in the state of the system, it is in the highest degree probable that they will have a special tendency to the induction of disease. It has long been a familiar fact that the human system is subjected to diseases varying in kind, intensity, and prevalency, with periods of the year. The cause of this has hitherto been sought for out of the system, viz., in the meteorological conditions of the season; but it will now be more proper to connect them with general states of the system due, doubtless, in a great degree, to those meteorological variations.

The dangers to which the body is peculiarly exposed by these variations in its vital actions are of two kinds.

1st. Those which result from the absolute degree of vital action in such a manner that the highest state may be regarded as indicating, or tending to, excess; and the lowest state a defect, or a tendency thereto, in the vital action—states known familiarly as sthenic and asthenic respectively. These dangers have reference to the *fixed* periods, and *will increase with the duration of those periods.*

2dly. Those which result from a want of sufficiently ready adaptation in the processes of the system to the sudden and varying influences from without; as, for example, those of temperature and food. These are peculiarly the dangers of the *variable* periods, and will be the *greatest at the commencement of the variation,* or when the changes are unusually violent or rapid.

If it be objected in reference to the former that the lower state of the functions is no evidence of lessened vitality or lessened amount of health, since with it there is a less amount of food taken and less exertion made, and thereby a due balance is still maintained, I would remark —the sense of languor and relaxation, with great heat and

lessened respiratory and other changes, lessened power of mental application and muscular exertion, special liability to the most destructive diseases at that period, the rapidity with which the inhabitants of hot climates succumb to disease, the increased liability to the attacks of disease when the system is enfeebled and the vital functions low, and *vice versâ;* and the greater amount of chemico-vital action in robust and healthy persons—all show that health in its two chief characteristics, viz., the power to resist external influences and the capability of the muscles to fulfil their functions, is lessened; and that, in fact, the term "asthenic" and the relative opposite term "sthenic" are applicable to this and the opposite state of the system. This appears to me to be a consideration of great consequence in the application of the effects of season to the production of disease, and if admitted will materially aid us in the determination of the vexed question as to the type of certain diseases.

I have found it of interest to compare these states of system at the different seasons and the dangers just pointed out with the mortality returns of a few diseases which have reference to this subject. It would have been more satisfactory if I could have obtained evidence of admitted value as to the true prevalence of disease; but, unfortunately, the admirable paper of Dr. Guy and the returns to the Board of Health still leave this important branch of knowledge too imperfect to permit me to rely upon it. In taking the mortality returns, however, whilst I shall lose some part of the value of the argument, I shall not import anything which is likely to lead to erroneous conclusions. I have selected the five years from 1850 to 1854, both inclusive, because, during that period, there was not such an amount of variation in the deaths as would disturb the averages—a circumstance which must have resulted if cholera or other epidemic years had been included.

Bearing in mind the four designations already given to the seasons in relation to the vital actions of the system,

viz., maximum, decreasing, minimum, and increasing, in their order, we find (in the accompanying table) that, in the average of the five years, the deaths from diarrhœa were 36·4 per cent. more numerous in the minimum period than would have occurred if they had been equally distributed over the year. Gastritis and enteritis—and to them may be added nephritis—diseases belonging to a class usually regarded as sthenic in its type, were each about 4 per cent. in excess at the same period. On the contrary, small-pox, measles, pericarditis, bronchitis, pneumonia, pleuritis, and peritonitis corresponded in intensity with the maximum period, and in the minimum period had so great a defect as from 4 to 14 per cent. Certain other diseases are not so restricted in their excess of mortality, but invade the variable periods with or without one of the fixed periods just mentioned. Thus, apoplexy is the most fatal in the increasing and maximum periods. Scarlatina has its greatest excess (12·5 per cent.) in the increasing period—that is, following the minimum period—and its greatest defect (8·3 per cent.) in the succeeding or maximum period; and in both respects typhus corresponds with it, whilst measles is as much in excess (6·4 per cent.) in the decreasing—that is, after the maximum period—as it is in defect (5·8 per cent.) in the succeeding or minimum period. Convulsions have their excess in the maximum, and defect in the minimum, periods.

In reference to epidemics—diseases admitted to be usually or entirely seasonal—it is to be remarked that yellow fever, and other virulent diseases of hot climates, are restricted to the minimum period. The plagues in London in 1593, 1603, 1625, and 1665, whatever was their nature, began soon after the minimum period had set in, attained the maximum in that period, and terminated in the first month of the increasing period. In like manner all the outbreaks of cholera in this country have invariably attained their great intensity in the minimum period; and although the disease existed, it made no ravages before or after it.

Influenza, on the other hand, when epidemic in 1847, had its maximum in the increasing and maximum periods.

Table showing the variation per cent. in the mortality from certain diseases in each quarter, on the average of the five years 1850-4, *from that which would have occurred if the deaths had been uniformly distributed over all the quarters of the year.*

Disease.	1st Quarter.	2d Quarter.	3d Quarter.	4th Quarter.
	Vital Changes.			
	Maximum.	Decreasing.	Minimum.	Increasing.
Diarrhœa . . .	— 15·2	— 14·5	+ 36·4	— 6·9
Enteritis . . .	— 1·7	+ 2·9	+ 4·	+ ·2
Gastritis . . .	— 2·4	+ 1·4	+ 4·4	— 4·6
Nephritis . . .	+ 2·3	— ·5	+ 3·4	— ·8
Peritonitis . .	+ ·7	+ 4·6	— 4·1	— 1·4
Pleuritis . . .	+ 5·	+ 5·	— 6·2	— ·3
Bronchitis . .	+ 12·9	— 1·9	— 14·	+ 2·8
Pneumonia . .	+ 4·8	+ 1·1	— 10·7	+ 6·7
Pericarditis . .	+ 4·5	+ ·3	— 6·4	+ 1·5
Cephalitis . . .	+ 1·6	+ ·5	.....	— 2·3
Convulsions . .	+ 2·7	— ·6	— 2·1	— ·2
Apoplexy . . .	+ 2·6	— 1·7	— 2·1	+ 1·2
Epilepsy . . .	+ 2·4	— 3·7	— 2·3	+ 3·
Smallpox . . .	+ 1·	+ 1·4	— 4·	+ 1·3
Measles . . .	— 1·1	+ 6·4	— 5·8	— ·1
Scarlatina . .	— 8·3	— 4·6	+ ·2	+ 12·5
Typhus . . .	— 2·1	— 2·	— ·5	+ 4·2

The relation which these diseases bear to each other in their understood sthenic and asthenic types, and with the states of the system now under illustration, is not without interest and importance. Thus there is the widest distinction between those affecting the bowels and the lungs. Cholera, diarrhœa, enteritis, gastritis, and epidemic fevers implicating the bowels, attain the maximum at the minimum period; whilst bronchitis, pneumonia, and pleuritis select the maximum period; and to the latter may be added the allied disease pericarditis, and also convulsions. There is also as marked a difference in the period when these diseases

begin to prevail. Scarlatina, typhus, apoplexy, epilepsy, convulsions, pericarditis, bronchitis, pneumonia, and pleuritis all increase at the increasing period; whilst measles, gastritis, and enteritis increase with the decreasing period. Cholera, the plague, diarrhœa, and yellow fever are so particularly restricted to the minimum period, that they can scarcely be said to receive their impetus in the variable periods; but if they do so, it is entirely as the vital changes are decreasing. The similarity under this head between scarlatina and typhus, and the dissimilarity between measles and scarlatina, well support the received opinion as to their several types. When these eruptive diseases greatly prevailed there was some difference, so that in the scarlatina of 1844 and 1848 there was a retrocession, and the most fatal periods were the minimum and increasing periods; and in 1845 measles were very prevalent at the same periods. Nephritis was more fatal at both variable periods, and so indeed was peritonitis, but the most so in the decreasing period.

Hence, as a general expression, it may be stated that at the fixed periods diseases of the bowels (minimum) and lungs (maximum) were the most fatal; whilst at the variable periods eruptive and kidney diseases prevailed in the different directions above mentioned.

The deaths from disease of the liver have also their maximum at the minimum period; but the difference with the season is not so marked in this country as to enable me to make use of this fact.

Deductions in reference to disease.—There are four deductions in view of this relation between prevalence and type of disease, and the state of the system, to which I desire to draw attention.

1. It is an admitted fact that the type of the same disease differs in different years, and we know that years vary in the character of their seasons, but into this question I do not purpose to enter. It is also admitted that the type of the same disease varies with the season, so that we look for inflammatory complications in one season and exhaustion in

the other. It is, I believe, admitted that, *cæteris paribus*, the same disease must be treated differently in different seasons. That certain diseases infest certain seasons chiefly or exclusively has just been shown, and universal experience has proved that diseases so restricted to the minimum period most rarely suffer depletory treatment. To sustain is the aim of the practitioner at that period. Hence, without entering into further detail, I think it may be affirmed that seasonal diseases depend more or less directly upon the degree of vital action existing at that period. This does not preclude the fashionable search after poisons as the cause of the disease; but it affords a rational and yearly occurring cause within the system for a yearly occurring disease or class of diseases.

The occurrence of a seasonal disease at other periods than its own proper season is easily understood, when we recollect the asthenic condition of so large a part of any community.

2. The type of a seasonal disease will vary according to the direction of the advancing season. Thus, an epidemic of measles occurring in the maximum or the decreasing period of the year, will not tend to inflammatory manifestations, as time advances, but the contrary, since the vital actions of the system are at that period diminishing. On the contrary, an epidemic of scarlatina at the increasing period might have at first presented a marked asthenic type, but with increase of the vital actions this is reduced, and sthenic complications become more apparent. So, it may be inferred, is the natural progress of any disease in which new cases arise over a lengthened period—the advancing type will be that of the advancing season. These facts are, I think, borne out in practice; but we must be careful not to form an opinion from an isolated case. Hence, in the treatment of seasonal, or indeed of any disease, it is important to bear in mind both the present and the advancing season.

3. Does not this discovery of the varying states of the human system in cycles help us to a rational explanation (at least in great part) of the cessation of seasonal diseases?

From our want of knowledge, we have been accustomed to say that the disease has worn itself out, or that, having carried off its victims, there is no more food for the destroyer, without our ever presuming that such explanations were satisfactory. But if an epidemic arises from, or only in, a certain state of system, it may increase as that condition of system increases; but it must decline when, by a further continuance, that condition of system is changing into its opposite. Thus, cholera or diarrhœa may appear in isolated cases at or before the beginning of June; but as that month advances, and the vital actions are decreasing, it bursts out with violence, and continues to increase until the middle of August, when the system has been attaining its lowest state of activity or vitality; but in September the deaths decline, and in October they cease—ceasing at a period when the system has assumed its upward tendency. Such, at least, has been the experience in our attacks of cholera, and such is now proved to be the progression in the vital actions. Only in this way can we account for the great excess of scarlatina in the fourth quarter, and its great defect in the following quarter. It is now well understood that, whatever inflammatory or febrile aspect it may assume, it is essentially a disease of the changing season, and is not sthenic in its character. Immediately the system has regained its maximum amount of vital power, it ceases to give development to scarlatina. As has been already shown, measles succeed to the maximum state, as scarlatina succeeded to the minimum state, and loses its sthenic character as the summer advances. Both diseases are most severe in the early part of the outbreak—the one by a tendency to exhaustion, the other to inflammation.

4. I venture also to suggest that this cyclical rotation in the variation of the vital powers is one of the explanations of the "Vis medicatrix naturæ." That there is a tendency implanted in the system to the cure of disease is undoubted; and as disease is commonly an exaggeration of the natural tendency of the system—that tendency varying as we have seen in direction and intensity in a defined and consecutive manner — a period must arrive when the

diseased state shall be placed under curative conditions, and the system which induced the disease shall become its own physician. This explanation may not be applicable to the cure of acute disease, but I deem it to be quite applicable to various states of the system not entirely dependent upon important structural changes.

In conclusion, it has been shown that whilst, in accordance with the statements of all observers, the respiratory function is ever varying, the changes pursue a definite cycle through the day and through the year. These two cycles are inseparably connected, for when that of the day is completed it does not always again start from the starting-point of the previous day, but assumes a new direction induced by the cycle of the seasons, so that the absolute quantities represented by the former are increased or decreased in a definite manner by the latter.

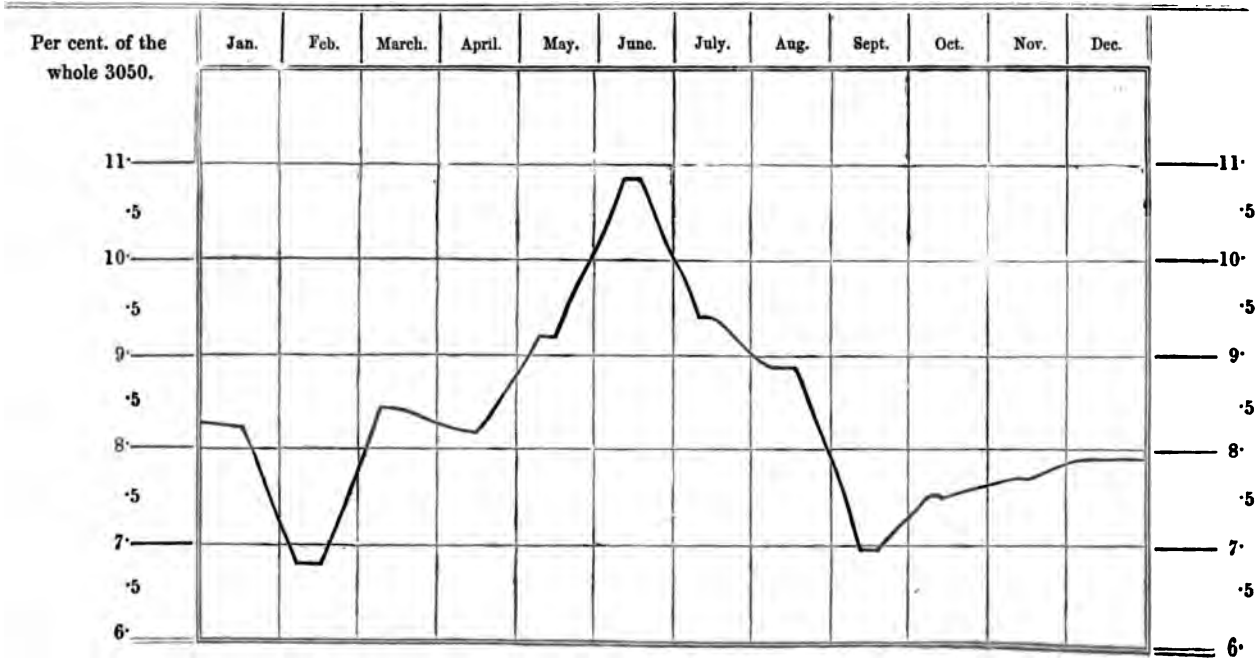
If there be truth in the observations now presented to the Society, we shall readily perceive that, however much the influence of seasons may induce disease, the succession of the seasons and their cyclical return is of the utmost value to man; and they give us another link in that great chain which binds the animal and vegetable creations together as one undivided whole. They also expose the folly of striving to maintain one unvarying season by artificial heat in winter and timid exposure in summer—an evil probably less prevalent now than in former years. They further show the propriety of freely exposing the body to the full influence of the ordinary changes of the seasons—a plan which, in the treatment of such a disease as phthisis, has effected an incalculable amount of good—as well as of protecting the system from the effect of seasons of undue severity or duration.

After a lengthened consideration of the subject, I am led to believe in the truthfulness of the views now given, and to hope that the demonstration of the seasonal changes of the system may lead to new ideas, and that some of the deductions now given contain the germ of fundamental truths.

Postscript.—*The influence of season on the viability of children.*—Since writing the foregoing paper, it has occurred to me to ascertain if the season of the year at which children are procreated or born affects their viability, or their subsequent liability to diseases of a type similar to that of the state of the human system existing at the season of their birth. By the kind permission of the Registrar-General and Dr. Farr, I have abstracted all the deaths of children dying in the northern district, from Newcastle to Kendall, in 1857 (a non-epidemic year), whose age in months was recorded with their death, and have thereby ascertained the period of their birth. The total number is 3050, nearly all of whom were under 1 year of age; and although the number is not very large, it is, in Dr. Farr's opinion, sufficient to reason upon. The result shows that of these children the largest per centage was born in June; and then in May, July, and August; whilst, after the latter month until January, the per centage was below the average. These months of diminished viability are those with which the human system has been shown to be in a declining or minimum state of vitality, and the effect is associated rather with the period of birth than the period of conception. The period is not that at which the greatest number of births occur. From this circumstance, as well as from the fact that the sexual appetites of animals have been so arranged that they are the most excited in the summer season (a period of year different from that of the birth of their offspring), it appears highly probable that the young differ in viability with the season of their birth, and therefore with the vital changes proceeding in their system at the period of their birth. This is also supported by the well-known fact of the influence of the health and constitution of the parent over the viability of the offspring.

I have commenced an inquiry as to the influence of the season of year at the time of birth over the tendency to the induction of certain classes of diseases, but it is still incomplete.

Diagram showing the month of birth of the children dying under one year of age. 1857.



A CONTRIBUTION

TO THE

STATISTICS OF CANCER,

COLLECTED FROM THE

CANCER RECORDS OF THE MIDDLESEX HOSPITAL.

BY

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COMMUNICATED BY

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THE object of the present communication is to lay before the Society the result of an examination of a somewhat large number of cases of cancer, which have been treated in the wards of the Middlesex Hospital.

The notes from which these results have been collected, were principally taken from cases treated during the years 1853, '54, '55, and '56. In order, however, to make them as satisfactory as possible in a statistical point of view, I have availed myself of the previous cancer records of the hospital. In many instances these notes are full, and the cases com-

plete, whilst many of the older records are less perfect, and can only be made use of for the deduction of a limited number of facts. The total number of cases noted is 520, with records of 173 post-mortem examinations. Out of these, 250 cases and 120 post-mortem examinations passed under my own observation.

These numbers are of sufficient magnitude, when the cases are considered in mass, to avoid the errors usually attending inferences drawn from the examination of a limited number of cases. On viewing separately, however, the examples of cancer in the various organs of the body, the numbers become so much reduced, that the conclusions derived from their consideration must be received with greater caution. It is not proposed in this communication to examine the cases upon all the numerous points in the pathology of the affection which might attract attention; but to select for investigation those subjects only, for the elucidation of which the cases appear to be more especially adapted.

Before proceeding further, it is necessary to explain what forms of disease have been comprehended within the limits of the present inquiry. The term Cancer has been used in a wide and extended sense, all the varieties of scirrhus, medullary, and epithelial growth having been included. The latter form of the disease presents so many points of distinction from the other two, that it seemed doubtful whether it would not be more desirable to reserve these cases for separate consideration. However, the numerous features of analogy between epithelial and true cancer, the occasional occurrence of transitional cases, and the association of the two forms in the same individual, led to a different conclusion. The results are, however, in most instances, so arranged, that the examples of epithelial cancer may easily be eliminated, if thought advisable.

Melanotic cancer has of course been included; and, for reasons hereafter to be stated, cases of "rodent ulcer" of the uterus have not been separated from the instances of true cancer in that organ. On the other hand, several

forms of disease sometimes considered to be cancerous have been excluded. Thus, cases of Lupus, forming a tolerably well-defined and distinct group, should be considered separately. The same may be said of the examples of Colloid.[1] Then, again, I believe Villous disease[2] to be simple and distinctive in its characters, and, although admitting that true Cancer may occasionally take on a villous or dendritic mode of growth, there is still no difficulty in distinguishing between simple Villous disease and dendritic Cancer.

PART I.

Seat of the primary cancer.—The table below exhibits the seat of the primary cancer in the cases forming the subject of the following statistics. It should be remarked that it does not show the relative liability of the different organs of the body to become affected with cancer, as there is an undue proportion of certain forms of the disease. This is doubtless owing to the circumstance that many of the class of hospital patients suffering from an external cancer, and knowing the nature of their complaint, naturally apply for admission to a cancer hospital, whilst those who are afflicted with the disease of the internal organs, and therefore ignorant of the cause of their illness, seek admittance into either of the general hospitals; and thus, in the wards of an hospital especially devoted to cancer, there is a preponderance of cases of the disease affecting the external parts of the body.

[1] See "The Structure and Nature of the so-called Colloid Cancer," by the Author. 'Med.-Chir. Trans.,' vol. xxxix.

[2] The examples of this form of disease which have occurred recently in the Hospital, are described in the 'Trans. Path. Soc.,' vols. vii and viii.

Table showing the seat of the primary cancer.

	Males.	Females.	Total.
Breast	1	191	192
Uterus	—	156	156
Labium, clitoris, &c.	—	13	13
Ovaries	—	2	2
Penis	6	—	6
Scrotum	2	—	2
Testicle	4	—	4
Lip, mouth, &c.	27	3	30
Tongue	9	5	14
Tonsil, palate, parotid, &c.	5	1	6
Œsophagus	2	1	3
Stomach, intestines, &c.	9	5	14
Rectum	4	7	11
Anus	4	1	5
Lungs	2	—	2
Liver	—	2	2
Kidneys	2	—	2
Lymphatic system	1	2	3
Thyroid body	—	1	1
Nose, face, scalp, &c.	10	9	19
Skin in other situations	5	5	10
Bones	9	6	15
Muscles, tendons, &c.	1	2	3
Eye	2	1	3
Moles, &c.	—	2	2
	105	415	520

Age.—In recording the ages of the cancer patients, care was taken to note the period at which the symptoms of the disease were first noticed; and in all that follows, the age spoken of is that of the patient at the time of the attack, and is not the age at which death took place. In considering this subject, it is convenient to separate the cases of cancer in each sex, and in the various organs. Among the female patients there were—

	Breast.	Uterus.	Other organs.	Total.
Under 10	—	—	—	—
From 10 to 20	—	—	1	1
„ 20 to 30	3	12	5	20
„ 30 to 40	31	34	12	77
„ 40 to 50	58	49	10	117
„ 50 to 60	40	19	13	72
„ 60 to 70	16	5	9	30
Over 70	5	—	2	7
	153	119	52	324

Among the cases of cancer of the female breast, the earliest age at which the disease commenced, was twenty-six. The oldest patient attacked with cancer of the breast was eighty-four. The average age at the time of attack of the 153 cases is 48·6 years.

In the cases of uterine cancer, the youngest was twenty-seven, the oldest sixty-five. The average age of the 119 cases is 43·28 years.

Among the instances of cancer in the female, neither the uterus nor the breast being the organ affected, the youngest was attacked at the age of fifteen (cancer of the hand), the oldest at the age of seventy-one. The average age of the 52 cases is fifty-three years.

In order to observe the effect of the climacteric on the numbers attacked with cancer, the decennial period from forty to fifty may be divided into two parts; the result is—

	Breast.	Uterus.	Other organs.	Total.
From 40 to 45	29	19	6	54
„ 45 to 50	29	30	4	63
Total from 40 to 50 ..	58	49	10	117

And with these numbers the following may be compared:

	Breast.	Uterus.	Other organs.	Total.
Under 45	63	65	24	152
Over 45	90	54	28	172
Total, all ages.. ..	153	119	52	324

It is thus seen that, although there is some difference in this respect between the cases of breast, and those of uterine cancer, there is, on the whole, no great difference in the gross numbers of those attacked before and after the age of forty-five. If, however, these numbers be compared with the total number of females living under and over the age of forty-five, it will be seen that the attacks are much more frequent after that age.

The ages of the male patients were—

	Of the lip.	Other organs.	Total.
Under 10	—	3	3
From 10 to 20 ..	—	—	—
„ 20 to 30 ..	1	6	7
„ 30 to 40 ..	—	3	3
„ 40 to 50 ..	5	12	17
„ 50 to 60 ..	4	7	11
„ 60 to 70 ..	1	14	15
Over 70	2	2	4
	13	47	60

Of the male patients attacked with cancer of the lip, the youngest was twenty-nine, the oldest seventy-nine. The average age of the 13 cases is 53·3 years.

Of those attacked with cancer of the other organs, the youngest was six months old (cancer of the eye), the oldest was eighty-two years (scirrhus of the mammilla). The average age of the 47 cases is forty-seven years.

Or, taking the cases in both sexes together—

	Males.	Females.	Total.
Under 10	3	—	3
From 10 to 20 ..	—	1	1
„ 20 to 30 ..	7	20	27
„ 30 to 40 ..	3	77	80
„ 40 to 50 ..	17	117	134
„ 50 to 60 ..	11	72	83
„ 60 to 70 ..	15	30	45
Over 70	4	7	11
	60	324	384

Of these the youngest was six months (male, encephaloid of

the eye), the oldest was eighty-four years (female, scirrhus of the breast). The average age of the 384 cases is 47·5 years.

From these tables it is seen that the age at which the patients were attacked, presented every possible variety, ranging from the earliest infancy to the extreme of old age. This great variation, however, is noticed only in a few isolated cases, the bulk being attacked after the middle period of life; the numbers increasing with age up to fifty; the highest proportion being in the ten years, forty to fifty.

It need hardly be observed that, in order to form a correct idea of the relative liability of the different ages to become attacked with the disease, it is necessary to compare these numbers with the relative numbers of persons living at these various ages.

The early years of life exhibit a remarkable freedom from the attacks of cancer; thus, under the age of twenty there were but four cases, their respective ages being six months, three, seven, and fifteen years. It is therefore seen that, during the period of life at which the nutritive powers are most fully exercised, and rapid growth is taking place, cancer hardly ever occurs. Directly, however, the frame is matured, growth having ceased, the liability to cancer shows itself; and this rapidly increases with advancing years till the maximum is reached, in the female about the climacteric period, and in the male soon after the full vigour of manhood is past.

There are some differences observed in the various groups into which the cases have been classed. For instance, among the cases of cancer in the female, there is a great difference in the age of those attacked with the disease in the breast, and in the uterus. The proportion of cases of cancer of the uterus between the ages of twenty and thirty is very large, as compared with that in the instance of cancer of the breast. Among the uterine cases, the number increases rapidly to the age of fifty, and then suddenly drops, no instance being met with after the age of sixty-five.

Among the instances of breast cancer, however, the number, increasing up to the age of fifty, only falls slightly to the age of sixty; and if the number is compared with the proportion of females living, the ratio of the attacks is as great between fifty and sixty as between forty and fifty. Again, the attacks in the case of the breast continue to a much later period of life. The same fact is also shown by comparing the average age of those attacked, this being 43·28 in the case of the uterus, and 48·6 years in the case of the breast.

It is also seen that the ages of females attacked with cancer in other parts, are somewhat greater than in the uterus or in the breast. This circumstance is partly owing to the fact that this group includes a considerable number of cases of epithelial cancer—a form of the disease which probably attacks the body somewhat later in life than the varieties of true cancer.

Among the male patients, it is noted that the variations in respect to age are greater than in the female. The first group, that of cancer of the lip (nearly all being epithelial), shows the average age of those attacked to be 53·3 years.

The second group includes cases of epithelial and true cancer, and exhibits very great variation in respect to the age of the patients.

The influence of marriage, pregnancy, the state of the catamenia, &c.

On taking the whole number of cases of cancer in the female, in which the civil state of the patient is recorded, it is found that there were—

Married	183
Widows	77
Single	55
Total . . .	315

which shows that rather over 17 per cent. were single

the remaining 83 per cent. either were, or had been married.

In order to exhibit more correctly the influence of marriage, &c., on the production of cancer, it is necessary to separate the cases of uterine disease from those of the affection attacking other organs.

1. Among the cases of cancer of the uterus there were—

Married	97
Widows	26
Single	12
Total	135

The fertility or barrenness of the patient is recorded in 100 cases. Of these, it is noted that 86 had borne children, and that 14 had not. Out of these 14, however, 3 had had one or more miscarriages, so that 11 only had never been pregnant.

The number of children borne by each of the 86 women varied from one to fifteen, the average number being 5·2.

As regards the interval between the last pregnancy and the attack with cancer, very great variation is observed. Among the 86 cases mentioned above, in 10 the attack was either during pregnancy or within a few months of it. The greatest interval recorded is twenty-seven years. The average interval of the whole 86 cases is 10·33 years.

Relation to the climacteric period.—In 85 cases in which the point is especially noted, the catamenia were still present, with greater or less regularity, in 62 cases, but they had ceased, either for many months, or for some years, in 23 cases. The longest interval noted between the cessation of the catamenia and the attack of cancer was seventeen years.

2. The cases of non-uterine cancer. Of these there were—

Married	86
Widows	51
Single	43
Total	180

The fertility or barrenness of the patient is recorded in 124 instances. Of these 98 had had children, of whom 26 had never borne a living infant; 9 had had one or more miscarriages; 17 had never been pregnant.

The number of children borne by each of the 98 women varied from one to eleven, the average proportion being 3·89.

As regards the interval between the last pregnancy and the attack, there is a similar amount of variation to that observed among the cases of uterine cancer. In 6 cases pregnancy either occurred during the existence of the cancer, or preceded it by a few months. The longest interval noted is thirty-seven years. The average interval among 80 cases in which the dates are recorded, is 10·2 years.

Relation to the climacteric period.—The catamenia had entirely ceased in 44 cases; they were still present in 50. The longest interval between the cessation of the catamenia and the attack of cancer was nineteen years.

These results show a great difference between the proportion of single women attacked with uterine cancer and those attacked with cancerous disease in other organs. In the instance of the uterus, the per centage of single women is under 9, whilst in the other organs it is over 23. So, again, a much smaller proportion of widows was attacked with cancer in the uterus than with similar disease in other parts of the body. So great a disproportion as the above can hardly depend upon accidental causes.

In a certain number of cases pregnancy took place while the patient was suffering from cancerous disease. The principal features of a few of these cases are given below.

Case 1.—J. C—, æt. 43, under the care of Mr. Moore, noticed that the left breast was indurated and nodulated. Six months after this discovery she became pregnant, and, after going her full time, was delivered of her second

child. The infant, however, only lived three weeks. After this the disease advanced; it attacked the other breast, and she died, aged 45, two years after first noticing the disease in the breast. No post-mortem examination.

Case 2.—E. R—, æt. 31, was attacked with cancer in the breast. Six months after this she miscarried. Subsequently she was operated on, and died aged 35.

Case 3.—C. N—, æt. 27, under the care of Mr. Shaw. Had had three children previously. In the second month of pregnancy the breast became hard and knobby. The disease advanced during pregnancy. She was confined at the full period, but no milk appeared in the breast. She died five months afterwards. Cancer was found in the internal organs.

Case 4.—H. F—, æt. 41, under the care of Mr. Arnott. Had had three children. When aged 38, attacked with scirrhus of the breast, with nodules in the skin around. When aged 40, she again became pregnant, and was delivered of a living child.

The general conclusions that these facts as to marriage, &c., point to, are—

1. That cancer attacks the married and single in different proportions according to the seat of the cancer, a much larger relative number of married women being attacked with uterine than with breast cancer.

2. That uterine cancer does not frequently occur in single women.

3. That when cancer attacks those who are, or have been married, it occurs among those who have had children and among the barren in about the same proportion as such individuals exist in the community. There is, therefore, no special proneness of cancer to attack the barren.

In this respect it may be observed, there is no difference between the cases of cancer of the uterus and of the other organs, the proportion of barren women being about the same in all.

4. That there are cases in which a patient already suffering from cancer has become pregnant, and that instances are also met with, in which the cancerous disease appears to have commenced during pregnancy.

5. As to the influence of the climacteric, it is observed that a larger proportion of persons affected with the uterine than with the other forms of cancer were attacked before the final cessation of the catamenia.

6. That as regards the fertility of those affected with cancer, the patients suffering from uterine cancer, as a rule, had had much larger families than those affected with the disease in other parts. This result is perhaps more remarkable, as the patients were attacked with uterine cancer at an earlier period of life than with the affection in other situations, and it is fair to believe that the occurrence of the disease in the uterus interfered with the subsequent childbearing of the patient.

If the average fruitfulness of all marriages be considered to be four (a number, it is believed, somewhat in excess of the real amount), it is then seen that the number of children borne by the patients suffering from uterine cancer (5·2) is 30 per cent. in excess of this number. On the other hand, the number of children produced by the other cancer patients (3·89) is 2¾ per cent. less than the average.

This comparison, however, is not perfectly just. There are two principal reasons why the average number of children borne by the cancer patients cannot be fairly compared with the average fruitfulness of marriage. In the first place, a few of the cancer patients had been married more than once, and some had borne children by each marriage. Secondly, many of the patients were attacked with cancer before the childbearing period was passed, and the number of children they might have had, was no doubt thereby diminished. These two

sources of error to a certain extent compensate each other, and, in the absence of more accurate data, the conclusions here given may be accepted as offering a fair approximation to the truth.

Duration of life in cases not operated on.—In order to determine the duration of life in patients suffering from cancer, those cases only are available, in which all the dates have been fully recorded; the number of cases thus complete is 225.

It should be premised that there is considerable difficulty in estimating the exact duration of the disease. In all internal cancers, the period given is less than the real duration, as it includes only the time during which the symptoms were present, the disease itself probably having existed for some months previously.

Among the female patients it is necessary to consider the duration of life—1st, in the case of the breast; 2dly, in that of the uterus; 3dly, in the other forms of external, or surgical, cancer; 4thly, in cases of internal cancer.

The number of available cases of cancer of the breast is 78. Among these the shortest duration of life was three months; the longest 172 months; the average being 32·25 months. This statement applies only to actual deaths from cancer; many other cases were noted as living, the disease having existed for a considerable time. In one only of these cases the period exceeds the maximum above given, the exception being a patient still living in the hospital, in whom the disease (scirrhus) had existed for twenty years (240 months). It is clear that a correct result would not be obtained if these cases were included, as the numbers above given include *all* patients *dying* of cancer, and are therefore likely to take in the proper proportion of examples of the acute and of the chronic forms of the disease.

Among 69 cases of uterine cancer the most rapid was two months, dating from the occurrence of the first symptoms. The longest duration was thirty-seven months. The average was 14·1 months.

This result shows a very striking difference in the average duration of life between those suffering from cancer of the breast, and those labouring under uterine cancer. It, however, exaggerates the real difference, as the duration of cases of cancer of the breast represents the whole period of the disease, whilst in the uterine cases the period is that of the duration of the symptoms, which, as a rule, is only the period of ulceration.

Among the 23 cases of external cancer in the female, (labium, skin, face, bones, &c.) the most rapid terminated fatally in four months (melanosis of the parotid); the longest duration was 113 months (epithelial cancer of the face). The average duration of the disease was 34·5 months.

Among the cases of internal cancer, (stomach, liver, ovaries, &c.) the most rapid was six months (cancer of the liver); the longest duration noted was twenty-eight months (scirrhus of the rectum); the average length of life after the first symptoms were observed being 21·3 months.

The cases of cancer in the male may be divided into two groups. In the first, the external cancer, (lip, face, penis, bones, &c.), the most rapid cases died in three months (one case of cancer of the femur, and one of cancer of the testis). The greatest duration of life was 124 months (epithelial cancer of the scrotum). The average of the 23 cases was 25·8 months.

Among the cases of internal cancer (stomach, lung, kidney, &c.), the most acute case died in two months (cancer of the stomach); the most prolonged case lived ninety-six months (encephaloid of the kidney). The average duration of life was thirteen months.

Or, taking the whole of the cases together, the average duration of the cancer in forty male patients was 20·4 months. In the 185 females it was twenty-five months. Of the total 225 cases of both sexes, it was 23·6 months, or rather less than two years.

As regards the variation in the duration of life dependent upon the situation of the cancer, the following gives the

periods in those organs, in which the number of examples is sufficiently great to form an average. (Both sexes.)

Breast . . .	32·25 months.
Uterus . . .	14·1 „
Stomach . . .	8·5 „
Rectum . . .	34· „
Lip, face, &c. . .	53· „
Penis . . .	34·5 „
Bones . . .	10· „
Labium . . .	29· „

Comparative duration of life in the different varieties of cancer.—To determine this point in a satisfactory mode it would be necessary to obtain the particulars of a considerable number of cases of the different forms of cancer attacking the same part of the body; but, as each part is affected by one form of cancer in particular, it is scarcely possible to obtain a sufficient number of instances to build a conclusion upon. It is necessary then to compare the different varieties of cancer, as they affect different parts of the body.

The cases of cancer of the breast may be taken as consisting entirely of examples of the scirrhous or medullary forms, the number of examples of the epithelial variety of the disease being too small to disturb the result. The average duration of life in these cases was 32·25 months. On the other hand, the instances of cancer of the lip may be looked upon as being epithelial (a very large majority being of this variety), and among these the duration of life was fifty-three months.

This fact indicates the great difference which exists in the rapidity of true, and of epithelial cancer. The two sets of cases are not of course perfectly comparable, in consequence of the different diseases which complicate the progress of cancer in these parts. The result, however, may be taken as indicating a difference in the duration of life, the causes of which are sufficiently obvious.

From what has been stated, it will be seen that it is useless to attempt to ascertain the difference in the

rate of progress in scirrhus and in medullary cancer, the line of demarcation between these two forms of the disease being so indistinctly marked. The present cases, however, bear out the received opinion that the softer forms of cancer are more rapid in their progress than true scirrhus; but there are among them several striking examples of encephaloid assuming a chronic form.

The effect of operation on the duration of life. — The foregoing results, derived from cases left to nature, should be compared with the duration of life in those cases, in which the disease had been removed by the knife. It will, however, be advisable to speak only of the cases of cancer of the breast, as the operations performed on other parts are not sufficiently numerous.

The total number of cases operated on is 57, and of these 6 were operated on twice, making a total of 63 operations. Out of these, 60 recovered from the effects of the operation, and 3 died, the mortality being 4·8 per cent.

Of the fatal cases, the first died thirteen days after the operation, the cause of death being pleuritis. In the second case, death also took place thirteen days after the operation, but was from pyæmia. The third died eighteen days after, from erysipelas, the patient also suffering from Bright's disease in an early stage.

Of the 60 cases in which recovery took place, the wound healed, or nearly healed, in 57 cases; in the remaining 3 cases the disease returned at once.

Cancer is recorded to have recurred locally sooner or later in 24 of these cases of recovery, this number being exclusive of the 3 in which the disease recurred at once. The latest period at which the local return of the disease was noted, was 108 months. The average period of the 24 cases was 14·8 months.

It is thus seen that, out of the 57 individuals operated on, 3 died of the operation, and in 27 the disease recurred in the part. Twenty-seven cases remain to be accounted for. Only 6, however, of these were traced for some time

subsequently to the operation, and they remained well when last seen. The periods of latest observation on 4 of these cases were seven, twenty-nine, thirty-six, and sixty-four months respectively.

Among the 24 cases in which the disease recurred locally, the earliest death was twelve months from the original appearance of the disease, the latest was 138 months; the average being 56·6 months. In order to make a just comparison with cases not operated on, it is necessary to include the 8 cases which died from the operation. This reduces the average duration of life, in cases operated on, to 58·2 months.

Or, again, taking the period at which death took place after the operation, it is found to vary from five to seventy-two months; the average duration of life after operation being 30·5 months.

It is thus seen that the patients operated on lived fifty-three months, whilst those on whom no operation was performed lived only thirty-two months, showing that the cases operated on lived twenty-one months longer than those left alone.

This result should not, however, be accepted without making due allowance for certain disturbing causes. In the first place, the cases submitted to operation are certainly more or less selected, and, as a rule, are to be looked upon as somewhat favorable forms of the disease, and thus the duration of life may be only apparently lengthened. Again, in the results just given, *all* the cases operated on have not been traced to their end; it is, however, right to infer that the numbers stated include a due proportion of cases in which the disease recurred quickly.

On the hereditary nature of cancer, and on the diseases affecting the families of cancerous patients.—The consideration of the question, whether cancer is hereditary, is a subject of great importance in its bearings upon the pathology of the disease. It is hardly necessary to observe that the relative degree of influence exercised by the local, and by

the constitutional causes which operate in the production of cancer, differs greatly in the various forms of the affection. Thus, in certain cases, as in soot cancers, the local irritation by itself appears to be sufficient to produce the disease, whilst in numerous other instances the cancer arises solely from the state of the constitution, there being an absence of local cause. It is thus seen that, although an apt state of constitution is considered necessary for the production of cancer, this apt state may arise in almost any individual, and that it may originate in families, in which the cancerous disease has not previously existed.

In order, therefore, to determine in what degree this state of system is hereditary, and how far acquired, it is necessary to ascertain whether cancer is more likely to occur among the members of a family in which some individual has already suffered from the disease, than in a family never previously affected.

It is obvious that the method of obtaining most satisfactory information as to the hereditary character of this, and, indeed, of any other affection would be, by noting in several families the diseases to which each member had been subject during life, and the cause of death of each deceased relative. However desirable it may be to investigate the subject according to this method, it is clear that the difficulties, especially among hospital patients, are so great as to prevent its being fully carried out. The number of instances in which any complete family history has been obtained, is too small to form the basis of conclusions. It is, therefore, proposed, in the present paper, to speak only of the occurrence of cancerous and of tubercular diseases in other members of the families, to which the cancer patient belonged.

The proof or refutation of the hereditary nature of cancer then rests on the solution of the question, "Does the proportion of instances of cancerous patients whose families had been previously affected with cancer, to those whose families had not been so, exceed the proportion of cancerous to non-cancerous families throughout the community?"

In obtaining a numerical solution to this question, many difficulties and numerous sources of inaccuracy present themselves, and the grounds of fallacy should be borne in mind.

The chief of these is perhaps the very imperfect knowledge which all, but more especially hospital patients possess as to the diseases to which their relatives have been subject. Even supposing a member of the patient's family to have died of cancer, and the fact to be distinctly known to the other relations, in the course of a few years the knowledge would become traditional, and probably the fact would cease to be remembered. The event of an individual dying of external cancer is, perhaps, less likely to be forgotten than if the death occurred from any less remarkable disease. If, however, the relative had died from internal cancer, the cause of the disease would probably be unknown, and thus the effect of these cases in increasing the proportion of cancerous relatives is nearly lost.

Compared with the foregoing, the inaccuracies dependent upon errors of diagnosis would scarcely produce any perceptible result, and as they would tell in both directions, causing some cancerous diseases to be considered non-malignant, and some simple diseases cancerous, the errors arising from this cause would nearly compensate each other.

All these sources of fallacy are reduced by taking a large number of cases; and, with a view of making the result as satisfactory as possible, the inquiries have been made with considerable care, the patients being not only asked, "Did any relative suffer from cancer?" but the age and the manner of death of each near relative was noted.

Out of the total number of 305 cases, in which it is stated whether cancer had, or had not been traced in any relative of the patient, it was found to have been present in the families of 34. One of these instances, however, was somewhat doubtful. Cancer was, therefore, traced

in 8$\frac{3}{4}$ per cent. of the total number of cases. The sex of the patients was as follows:

	Males.	Females.	Total.
No. of cases ..	42 ..	263 ..	305
Cancer traced in ..	4 ..	30 ..	34

The patients who remembered to have had a cancerous relative were—

	Males.	Females.	Total.
Breast	— ..	17 ..	17
Uterus	— ..	8 ..	8
Vagina	— ..	1 ..	1
Rectum	— ..	1 ..	1
Anus	— ..	1 ..	1
Scalp, face, &c. ..	— ..	2 ..	2
Lip	1 ..	— ..	1
Bones of face ..	1 ..	— ..	1
Antrum	1 ..	— ..	1
Stomach	1 ..	— ..	1
	—	—	—
	4	30	34

Out of the 34 in which cancer was traced, in 27 it existed in only one relative of the patient, and in the remaining 7 more than one relation was affected with the disease. The degree of relationship of the cancerous subjects was as follows:[1]

In the mother or father . . .	13
„ grandmother	2
„ great aunt	1
„ aunt or uncle	10
„ cousin	6
„ brother or sister	8
	—
	40

[1] In this table the cases in which more than one relation was cancerous, are included, the total number therefore exceeds the number of patients who had relations affected with the disease.

The hereditary taints in question may be arranged as follows:

On the father's side	7
„ mother's side	19
On both sides	1
Not noted	5
	32

A different degree of importance is to be attached to the presence of cancer in relations of the various degrees of kinship. Thus, its presence in a brother or sister (except that here other causes interfere with the deduction) should be an argument of the strongest force, as brothers and sisters possess constitutions nearly identical, or, as it is said, are of the same blood. Its presence in one of the parents is an argument of less force, for supposing the father and mother to contribute their constitutional peculiarities in equal proportions, the child and either parent have but half their constitutions identical. In like manner the presence of the disease in a cousin proves still less, as they have but one fourth of their constitutional peculiarities in common. And so on with any other relationship, the force of the argument to be derived from an example of a cancerous relative varying in proportion to the amount of constitution the two possess in common.

The cancerous relations of the cancer patients were thus affected—

Breast	14 cases	Eye	1 case
Uterus	5 „	Liver	1 „
Labium	1 case	Leg	1 „
Lip	2 cases	Not noted	6 cases
Throat	2 „		

The instances in which cancer affected more than one relative of the patient, were—

Case 5.—A. S—, female, æt. 44. Cancer of the breast. Her father's sister and a cousin died of cancer.

Case 6.—S. S—, female, æt. 28. Cancer of the nose. Her uncle, and a cousin died of cancer.

Case 7.—C. M—, female, æt. 59. Cancer of scalp. Her mother, and a sister died of cancer.

Case 8.—D. F—, female, æt. 32, died of scirrhus of the cervix uteri. Her mother died, aged fifty-two, in the Middlesex Hospital, some years before, of cancer of the uterus. A maternal aunt had died of cancer of the breast.

Case 9.—E. L—, æt. 29, had cancer of the uterus. Her father's mother died aged, of cancer of the anus. A maternal aunt had been operated on for what was considered cancer of the breast.

Case 10.—S. H—, married. Was born in 1803. A cancerous tumour formed in the left breast in 1846, and was excised a month or two afterwards by Mr. Arnott. The tumour again returned at the end of 1855, and was excised in the following April by Mr. Shaw. The patient recovered, and remains well.

The patient's family consisted of the following members:

1. The eldest being the patient herself.
2. A brother who died of phthisis in 1847.
3. A single sister born in 1811, died in 1844 of cancer of the breast. Had not been operated on.
4. A single sister born in 1817, died in 1847 of cancer of the breast. Had not been operated on.
5. A married sister had a scirrhus of the breast removed in 1845, and again in 1856; is still living.
6. A single sister had a cancer of the breast removed in 1852, and died of erysipelas after the operation.
7. A married sister, who remains at the present time in good health.

The mother of this family died of cancer of the breast in 1829. She had three sisters, two of whom died of phthisis, and one of dropsy.

The father died from an accident. His brothers and sisters were long-lived and healthy.

The patient's father's father died of hæmoptysis, and his father lived to the age of 101.

In all the instances in which cancer affected any members of this family, the left breast was the seat of the disease.

The occurrence of phthisis in the families of cancer patients.—It is noted in 130 cases whether phthisis existed, or not, in the family of the patient. The question was answered in the affirmative in 48 instances.

Out of these 48 cases in which there was a history of phthisis in some member of the family, 43 were females and 5 were males.

The form of the disease in the 43 females was—

Breast . .	18	Skin . .	2
Uterus . .	19	Tongue . .	2
Ovary . .	1	Hand . .	1

Among the 5 males the disease existed in the following organs—femur, lip, skin, anus, soft palate (there being one example of each).

Again, out of the 48 cases, in 20 instances more than one relative was affected with phthisis, and in some cases as many as 6 relatives had been affected.

The relations suffering from phthisis in these cases were—

Father or mother	32
(Both being affected in 2 cases.)	
Brothers or sisters	27
(In 11 cases more than one brother or sister affected.)	
Grandmother or grandfather . . .	2
Children	7
(In 2 cases more than one child affected.)	
	68

In the foregoing, only cases of pulmonary consumption have been included, instances of the other forms of tubercular disease, such as scrofula, hydrocephalus, tabes, &c., being neglected, as offering numerous sources of fallacy.

It is thus seen that in 8¾ per cent., or in about one eleventh of all cases affected with cancer, the same disease had existed in other members of the family.

From this fact taken by itself little can be inferred, and its value as an argument for or against the hereditary nature of cancer, must rest on the solution of the question, "Does this ratio exceed the proportion of hospital patients suffering from other diseases, and taken indiscriminately, who would remember to have lost a relative from cancer?" Without possessing sufficiently extensive information upon this subject, I can only state my belief that the above number (8¾ per cent.) is not greatly in excess of the proportion of hospital patients who remember to have had cancerous relatives. The circumstance, therefore, taken alone is not to be regarded as convincing evidence of the hereditary nature of cancer.

A much stronger reason, however, for believing that the disease may be transmitted from parent to child, is derived from the fact that, in 6 instances out of 34 in which cancer existed in the family, more than one member had been affected. In 5 instances 2 members had suffered from the disease, and in the sixth case 5 relatives were cancerous.

Out of the total number of patients who had cancerous relatives, (34) it might be expected that the same ratio would again hold true, and that 2, or even 3, of these patients would have more than one relative affected with cancer. The proportion, however, which is found to exist being so much larger than that which would be accounted for upon this principle, it is clear that other causes beyond mere accidental coincidence must come into operation in producing the result.

The last quoted case by itself forms as strong an argument in favour of the hereditary nature of the disease as an individual case possibly could, and taken in connexion with the other facts, it constitutes all but conclusive evidence that cancer is, to a certain extent at least, hereditary.

It is also seen that phthisis and cancer coexist with great frequency in different members of the same family. Phthisis

was present in 48 out of 130 cases, or in nearly 37 per cent. The disease, moreover, existed in a brother or sister of the cancerous patient in 27 cases, or in more than 20 per cent. It need not be discussed here, whether the fact indicates simply that the two diatheses may coexist in the same family, or whether it tends to show that the one diathesis may be a modification of the other.

It might have been expected that a larger proportion of relatives should have been affected with phthisis than with cancer, because the former disease is so much more common, and also from the fact that patients die from it at a much earlier age, and that, therefore, a larger number of relatives may be included. Owing to this latter circumstance it is evident that, in obtaining the family history, all the instances of consumption in the brothers and sisters of the patient would be recorded, as they would probably have passed the age liable to the attacks of this malady. In the case of cancer, however, it is equally likely that the brothers and sisters should be attacked after as before the patient, and in the former case these instances would not be included.

The great differences in the age of those attacked should be borne in mind in estimating the relative influence of the hereditary causes in cancer and in phthisis. The fact of several members of the same family, living under precisely the same conditions as to residence, food, habits, &c., being attacked with phthisis, is by no means a very convincing argument in favour of the hereditary nature of the disease. On the other hand, several members of a family being attacked with cancer at a time of life when they would be living under different conditions, in different places, and following separate occupations, is an argument of much greater force in favour of the hereditary transmission of cancer.

PART II.

An analysis of the records of the post-mortem examinations of cancer patients.

The number of post-mortem examinations available for the purpose is 173. The following table exhibits the seat of the primary cancer in each instance:

	Males.	Females.
Breast	—	61
Uterus	—	44
Vagina	—	2
Ovaries	—	2
Clitoris, labia, &c.	—	2
Penis	2	—
Scrotum	2	—
Testicle	2	—
Lip	7	—
Skin in various situations	6	3
Alveolus	2	—
Tongue	1	—
Throat, palate	1	3
Œsophagus	1	—
Stomach	5	—
Peritoneum	—	1
Intestines	1	—
Rectum	2	4
Anus	—	1
Liver	—	3
Lung	1	—
Lymphatic glands	1	1
Parotid gland	1	1
Thyroid gland	—	1
Bones	4	2
Muscles	—	2
Eye	1	—
Total	40	133

The first subject which presents itself in analysing these

cases, is that of the dissemination of cancer structures in different parts of the body. It is well known that the multiplication of the disease is observed only in a certain proportion, and not in all examples of the affection. In one group of cases the cancer is strictly local, it does not even affect the lymphatic glands of the part. In a second group there is, in addition to the local affection, disease in the lymphatics, the internal viscera, however, being unaffected. In a third group the whole system appears contaminated, and the disease is scattered among the various organs of the body. The foregoing cases may therefore be arranged—

Disease of the part only . . .	29
Affecting also the lymphatic glands . .	44
State of lymphatic glands doubtful . .	12
Disease affecting the system . . .	88
	173

In the above it should be remembered that the affection is considered local, when there is but one mass of diseased structure. For instance, there are cases of scirrhus of the breast passing into the chest, and involving the mediastinum and pleura, there being no disease elsewhere. These are classed under local cancers, as there was but one mass of diseased growth in the body.

In order to judge of the indications of the above table, it is necessary to bear in mind the proportion of the different varieties of cancer which enter into the above. They are—

Scirrhus	103
Medullary (including melanosis) .	41
Epithelial (including rodent ulcer) .	29
	173

The distribution of the secondary cancers in the various organs of the body is shown in the following table:

	I. Breast.	II. Uterus.	III. Epithelial, of various organs.	IV. True Cancer, various organs.	V. Total.
1. The disease not extending beyond the lymphatic glands	13	34	12	24	83
2. Affecting lungs, &c., but liver healthy	5	—	—	2	7
3. Affecting liver, &c., but the lungs healthy . . .	25	5	1	14	45
4. Involving both lungs and liver	8	2	1	4	15
5. Involving other parts, but both lungs and liver healthy .	10	3	1	9	23
	61	44	15	53	173

This table exhibits the comparative frequency with which the various internal organs are affected in the different forms of cancer. In the first and fourth columns all the cases are examples of true cancer, those in the first being almost entirely scirrhous, but those in the fourth being partly scirrhous and partly medullary. Among the cases of uterine cancer there are a few epithelial, and some cases of rodent ulcer, the rest being examples of true cancer. The third column consists entirely of epithelial cancer of the external organs.

I. *Cancer of the breast.*

Among the 61 cases of cancer of the breast the state of the lymphatic glands was healthy in 8; more or less diseased in 53.

The cancerous disease found among the various organs of the body in these cases was as follows:

In the lungs	13
„ liver	33
„ kidney	1
„ spleen	1
„ pleuræ	17

In the pericardium	9
„ peritoneum	9
„ arachnoid	2
Extensive cancer in the bones	6
In the supra-renal capsules	2
In the other breast	9
Very numerous nodules beneath skin	8

The mode in which the various secondary cancers were associated together, is shown in the table before given.

The following non-cancerous diseases were found in the bodies of those affected with cancer of the breast:

Pleuritis, with effusion	21
Pleuritis and pericarditis	3
Pericarditis	1
Bright's disease	2
Abscess near the cancer	2
Pyæmia	1
Tubercle, phthisis	2
Caries of the spine	1
Cystic disease of the ovary	1
Fibrous tumour of the uterus	3
Cirrhosis of the liver	1
Apoplectic cysts in the brain	1
Disease of the mitral valve	1

II. *Cancer of the uterus.*

There are 44 examples of uterine cancer. This number, however, includes several examples of the "rodent ulcer." The principal reason for classing these cases with the examples of true cancer of the uterus is the extreme difficulty, if not impossibility, of distinguishing between them. This difficulty is no doubt owing to the fact, that ulceration commences very early in cases of cancer of the uterus, so that by the time the life of the patient is destroyed, nearly all the cancerous tissue is destroyed, and therefore the remaining ulcer can hardly be distinguished from the rodent ulcer. In some instances even of apparent rodent ulcer, the

nature of the disease is proved by the presence of cancer in other organs. This condition was exhibited in a recent case, in which the cervix uteri was destroyed by what appeared to be a rodent ulcer, no cancerous tissue being detected about its edges, but in connexion with the uterus the two ovaries were converted into large cancerous tumours.

The following secondary cancers were found in these patients:

In the lungs	1
„ liver	7
„ kidney	1
„ pleura	2
„ pericardium	2
„ peritoneum	1
„ ovaries	1
„ intestines	1

The lymphatic system was affected as follows:

The disease strictly local	15
Affecting only the part and lymphatic glands, &c.	19
Affecting other organs	10

The non-cancerous diseases found in the bodies of those suffering from uterine cancer, were as follows:

Vesical fistula	10	Fatty liver	1
Rectal fistula	2	Hydatids in liver	1
Ileus	1	Rickets	1
Pyelitis	1	Pleuro-pneumonia	2
Abscess of the kidney	1	Fibrous tumour of uterus	5
Tubercle in the lung	4	Cystic ovary	2
Bright's disease	4	Arachnoid hæmorrhage	1

III. *Epithelial cancer.*

Among the 15 instances of epithelial cancer, the state of the lymphatic system was quite healthy in 4; cancerous in 8; extensive disease of the lymphatic system in 3.

That is to say, the lymphatics were quite healthy in 4

cases, and cancerous in 11; but in 8 out of these 11 instances nearly the whole of the lymphatics of the body were affected with epithelial cancer. In one or two of these cases an appearance of cancer of the lung was presented, owing to a lymphatic gland imbedded in the lung-tissue being affected with epithelial cancer. It is obvious, however, that it would not be just to place these cases as examples of epithelial cancer of the lung.

It will be seen in the table before given, that the disease was confined to the lymphatics in 11 out of the 15 cases; in the remaining 4 cases cancerous disease was found in other parts of the body. There is, however, only one genuine example of two distinct epithelial cancers in one individual. In the other 3 cases the secondary tumours were examples of another form of the disease. The following are the chief features of the case in which there were two distinct epithelial tumours.

Case 11.—*Epithelial cancer of the leg and of base of tongue.*—M. S—, male, æt. 63, a wood-sawyer, married; a tall man of slight make. Twelve months before he came under observation, a tubercle formed beneath the skin on the inner aspect of the left leg. When he first discovered it, it was not larger than a pin's head; but having scratched the surface, it became sore, and has never healed since. When admitted in November, 1853, under the care of Mr. Henry, there was a tumour the form and size of half an egg; the surface covered with fine granulations, and a fissure separating it from the skin around. Soon after admission he began to complain of a sore throat, and in the course of a short time an ulcer was discovered at the base of the tongue, which soon extended into the tonsil. The glands beneath the jaw became enlarged. After this, the sore on the leg remained in a stationary condition, the throat gradually became worse, the ulceration extending and producing great difficulty in swallowing food. An opening made its appearance over the glands beneath the jaw. He gradually sank, and died on May 12th, 1854.

In the post-mortem examination, the tumour in the leg and that at the base of the tongue exhibited the usual characters of epithelial cancer, both to the unassisted eye and beneath the microscope. The glands beneath the jaw were affected by the disease, but the femoral glands were quite healthy.

The following is one of the cases, in which soft cancer became developed as secondary tumours to epithelial cancer.

CASE 12.—*Old-standing epithelial cancer of the scrotum; extensive disease of the lymphatic system; medullary cancer in the liver; secondary syphilis; caries of the spine.*—James G—, æt. 30, a chimney-sweeper, married, was admitted into the hospital October 9th, 1855, under the care of Mr. De Morgan. In the spring of 1850, a tumour formed in the scrotum, over which the skin became ulcerated, but healed at the end of twelve months. A year after this another similar tumour formed, and remained ulcerated about the same time as the first, and healed, and shortly afterwards again became ulcerated. A twelvemonth before admission, he had venereal disease, which is described to have been without sores. Six months before he came to the hospital, the glands in the groin became enlarged. When admitted, there was a portion of skin in the scrotum, the size of a shilling, which was hard and thickened, the surface over being abraded. Close at the side of this, is the cicatrix of the original lump, which is perfectly healthy. In the left groin an irregular sore, with thin discharge, the skin being excavated around. A similar opening in the right groin. On the face a copious eruption of red elevated tubercles.

After this, the openings in both groins extended, and discharged more profusely. He became rapidly emaciated, and suffered from intense neuralgia in the thigh. The eruption gradually faded, and he died in January, 1856.

At the post-mortem examination, the lymphatics of the

pelvis and lumbar region were affected with extensive cancerous disease, which presented the same appearances as the tumour in the scrotum. The only other cancer was a tumour in the liver; the structure of this was distinct from that of the scrotum, and did not exhibit the characteristics of epithelial cancer. Extensive carious disease of the spine was also discovered.

The relative frequency of the occurrence of secondary Tumours in the different forms of Cancer.

From the foregoing, it will be seen that there is a great disproportion as to the number of instances in which secondary tumours occur in the various forms of cancer. Thus, in cancer of the breast there were secondary tumours in 79 per cent. of the total number of cases; in true cancer of other organs in 54 per cent.; in uterine cancer in 23 per cent.; and in epithelial cancer in 20 per cent.

The table before given (page 140) moreover exhibits the different modes in which the secondary tumours were associated in the same body. In each form of the disease nearly every possible combination of the secondary tumours was observed, with the exception that among the cases of cancer of the uterus there was no example of the disease attacking the lungs, the liver remaining healthy.

The multiplication and dissemination of Cancer.—Without attempting to discuss fully the various means by which the multiplication and dissemination of cancer occurs, a few words may be said on the bearing of the present cases upon this subject. There appear to be three principal modes in which a cancerous growth may be multiplied.

1. By the development of cancerous nodules in the immediate neighbourhood of the original tumour.

2. By the growth of cancerous disease in the lymphatic glands of the part.

3. By the formation of tumours in distant parts of the body, and more especially in the lungs and in the liver.

By the first method, the development of cancerous nodules probably depends on the infiltration of a blastema endowed with the property of cancerous development in the meshes of areolar tissue near the tumour. By the second mode, the growth of cancer in the lymphatic glands may be supposed to arise from the absorption of cancer-germs, which either become developed, or excite the formation of cancer-cells in the lymphatic glands. In a few instances, however (and chiefly, in cases of epithelial cancer), the disease in the glands may be traced to have extended from the part originally affected, along the lymphatic vessels. In the third instance, the growth of tumours in distant parts of the body, the blood itself is contaminated, and carries the cancer-germs to the various organs of the body.

It is probable that the multiplication of cancer takes place by each of these methods. The first is necessary to explain the occurrence of the numerous nodules which are found so frequently in the parts adjacent to a cancer, as in cases of scirrhus of the breast, and in some instances of epithelial cancer. Then, again, there are several instances in which secondary tumours were discovered, but both the lungs and the liver escaped the attacks of the disease. Thus, in 22 out of 172 cases there were secondary tumours, the lungs and liver remaining healthy. So, also, in 10 out of 61 examples of cancer of the breast, the same fact was observed. In a large proportion of these cases, the secondary tumours were not very distant from the original cancer; but in a few examples, the other viscera (spleen, kidney, &c.) were affected. In all these instances, the multiplication of the cancer appears to have depended on local, rather than on constitutional causes; for if the system itself were much involved, the viscera would not be more likely to escape the ravages of the disease, than the parts near the original tumour.

The blood becomes secondarily contaminated by the agency of the lymphatics, or by the blood-vessels. In the

lymphatics, cancerous material was frequently observed; but in the veins, although clots were often noticed as having a somewhat cancerous appearance, yet, on careful examination, the structure of cancer could not be demonstrated.

In this enumeration, I have not alluded to the theory, which supposes that the formation of secondary tumours depends solely on the state of the system, and that there is no subsequent contamination of the vital fluids; that, in short, the same state of blood which gave rise to the first tumour, produces the second. If this theory were true, secondary tumours should form in the favourite seats of cancer, and not, as occurs in most cases, in the lungs and liver. Thus, for instance, a patient suffering from scirrhus of the breast should be attacked with the disease in the uterus, another with cancer of the eye should have the secondary affection in the stomach. Such a distribution of the disease, however, does not occur; the foregoing cases do not afford a single example of what might be called two primary cancers, the secondary tumours in nearly every instance being arranged in a determinate method.

The diseases affecting cancerous patients.

The inquiry into the laws which govern the association of various diseases in the same individual, is one of the most interesting branches of pathology, and it is of greater importance in a disease of obscure origin, like cancer, as by such an inquiry some light might be expected to be thrown on the causes of the affection.

The non-cancerous diseases which were found in the bodies of the cancer patients, may be placed in two groups.

1st. Those diseases which were obviously brought about, or produced by the cancer.

2d. Those which appear independent of it.

Among the first group we find—

Pleuritis	25	Pyæmia	1
Pleuropneumonia	9	Abscess near the cancer	2
Vesical fistula	11	Pyelitis	1
Rectal fistula	2	Abscess of kidney	1
Pericarditis	6	Œdema of the glottis	1
Extensive phlebitis	1	Ileus	2

All the foregoing were clearly produced by the cancerous disease. The second group is of greater interest, as showing some of the constitutional diseases which may affect cancer patients.

Tubercle in lungs	15	General dropsy	3
Caries of the spine	2	Diseased mitral valve	3
Rickets	1	Fatty degeneration of heart	2
Active syphilis	1	Apoplectic cysts	1
Bright's disease	10	Congestion of brain	1
Cirrhosis of the liver	2	Arachnoid hæmorrhage	1
Waxy liver	2	Plugging of cerebral arteries	1
Hydatid of liver	1	Fibrous tumour in uterus	9
Enlarged spleen	1	Cystic diseased ovary	3

From the above, it is seen that nearly any of the more common diseases may co-exist with cancer. Rheumatism and gout appear to form an exception to this, none of the foregoing cases affording an example of the concurrence of either of these diseases with cancer.

The diseases which are less frequently, as well as those which are most commonly associated with cancer, should be noted. Cardiac affections are perhaps the most remarkable in this point of view, it being very rare to meet with advanced cardiac disease in those dying of cancer. Among the post-mortem examinations, there are only three cases of marked valvular disease. Hypertrophy of the heart is extremely rare among cancer patients, the present cases not affording a single example of decided hypertrophy; and in connexion with this circumstance, it should be remembered that many patients suffered from Bright's disease. The explanation of this somewhat remarkable absence of examples of hypertrophy, probably rests in the fact, that among

cancer patients the quantity of blood in the body is reduced to such an extent, as to require but little force to propel it along the vessels. The tendency of the heart to become hypertrophied, which is so usually met with in patients suffering from Bright's disease, would thus be counteracted.

The concurrence of Tubercle and Cancer.

Tubercle was found in the bodies of cancer patients in the following cases:

Case 13.—T. P—, male, æt. 59, dying of encephaloid of the stomach, under the care of Dr. Hawkins. A large nodule of calcareous tubercle in the apex of the lung.

Case 14.—M. H—, female, æt. 48, dying of scirrhus of the breast, under the care of Mr. De Morgan. A large quantity of recent tubercle in upper lobes of both lungs, with large cavities. (See Museum of Middlesex Hospital.)

Case 15.—W. B—, male, æt. 38. Cancer of the tongue, under the care of Mr. Moore. In the lung, a cavity of the size of a small orange: infiltrating semi-transparent tubercle around. Old tubercle in the mesenteric glands. Ulceration of the colon.

Case 16.—T. B—, male, æt. 53, under Mr. De Morgan. Medullary cancer of the head of the femur. A nodule of cheesy tubercle was found in the upper lobe of the lung.

Case 17.—A. E—, male, æt. 69, under Dr. Seth Thompson. Firm medullary cancer of the root of the lung, there being cancerous disease also in the kidney and in the mesenteric glands. In both apices there was cretaceous and recent tubercle. In the apex of one of the lungs there was a cavity, of the size of an orange.

Case 18.—A. R—, æt. 54, under Mr. Shaw, scirrhus of the uterus. Several deep-seated calcareous nodules were found in the right lung.

Case 19.—M. R—, female, under Dr. Stewart. Medullary cancer of the ovaries, there being also cancer of the liver. In this instance there was recent tubercle in both apices.

Case 20.—G. O—, male, æt. 50, under Dr. Hawkins. Encephaloid cancer of the stomach. In both apices miliary and crude tubercle, a small cavity in the right apex.

Case 21.—E. W—, æt. 41, under Mr. Moore. Medullary cancer of the uterus and glands. A large quantity of miliary and crude tubercle in both apices. A small cavity in the right.

Case 22.—J. S—, æt. 57, under Mr. Shaw. Firm medullary cancer of the breast. A cicatrix was found in the left apex. There was also caries of the vertebræ.

Case 23.—W. N—, under Mr. Moore. Epithelial cancer of the lip, and of the lymphatic glands. Large cicatrices, with old tubercle (partly cretaceous) in both apices.

Case 24.—S. C—, female, æt. 48, under Mr. De Morgan. Scirrhus of the vagina, and of the lymphatic glands. Recent tubercle and cicatrices were found in both lungs.

Case 25.—W. H—, male, æt. 64, under Dr. Stewart. Encephaloid of the stomach. A few tubercles were found in the apices.

Case 26.—J. C—, male, æt. 48, under Dr. Seth Thompson. Scirrhus of the pylorus, with cancer in the lymphatic glands, liver, peritoneum, &c. There were cretaceous tubercles in both lungs. In this case the liver was affected with cirrhosis.

Case 27.—R. S—, female, æt. 65, under Mr. Shaw. Encephaloid of the face, there being also similar disease in the kidney. In this case there were recent tubercles in the apices of both lungs.

The cases in which tubercle was found, were therefore as follows:

Epithelial cancer	1
Scirrhus	5
Soft cancer	9

And the form in which the tubercle was formed was:

Cretaceous	4
Crude and miliary . . .	6
With cavities	5

The disease was therefore of recent date in 11 cases, whilst it was of old standing in 4.

The immediate causes of death among cancerous patients. Influence of cachexia, &c.

In watching the progress of cancer among the patients of the hospital, it is remarkable to find that the cachexia is closely proportional to the amount of hæmorrhage, discharge, and pain. In cases in which there is but little hæmorrhage, and a small amount of discharge, the cachexia is hardly obvious, and this is usually observed, even where the cancerous tumours have attained great magnitude. It sometimes happens that the cachexia becomes well marked, even where there is but little hæmorrhage, or discharge; but, in these cases, the cancer is usually found to have involved some important internal organ, and to have interfered with some vital function.

On the other hand, in those patients with whom there is profuse discharge, and frequent attacks of hæmorrhage, the wasted sallow visage of advanced cancerous disease becomes obvious at an early stage of the complaint. In no class of cases is the cachexia more pronounced than in uterine cancer, or even in the instances of rodent ulcer of the uterus. The amount of cachexia is therefore wholly independent of the size of the cancerous tumour; it is often absent in cases of enormous growths, and may be present where there is but little cancerous tissue.

Moreover, in instances of benign tumours, which pass

on to ulceration, a form of cachexia often makes its appearance. Thus, in a recent case, a woman suffering from ulcerated proliferous cystic tumour of the breast presented a condition of extreme cachexia.

From these conditions I cannot but come to the conclusion, that the presence or absence of cachexia is valueless as an aid to diagnosis. It appears to be the result of the local disease, and is not to be regarded as evidence of a state of system which leads to the production of cancer.

On analysing the results of the post-mortem examinations, it does not appear that any patient has actually died of cancerous cachexia, the death in each instance having arisen from an amount of disease which would have been quite sufficient to kill, even had it been of a benign nature, and not from its peculiar character affecting the composition of the blood, or the powers of the system. The immediate cause of death is shown in the catalogue of diseases induced by the cancer. Thus, in 61 cases of cancer of the breast, pleuritis with effusion was present in 24 cases, and in this number only well-marked instances were included. Among the uterine cases, the frequency of the occurrence of fistula is scarcely less than that of pleuritis in cases of cancer of the breast. So again, cancer in other situations induces secondary diseases, which in a large proportion of cases are the immediate causes of death.

The more important conclusions to which the foregoing analysis leads, may be thus arranged:

1. In respect to age, it was observed that nearly all the patients had arrived at maturity, four only being attacked under the age of twenty years. The average age of those attacked with the disease in the uterus was forty-three and a quarter years; in the breast forty-eight and a half years. The patients with epithelial cancer were somewhat older than those with uterine, or breast cancer.

2. Childbearing appears as one of the predisposing causes to the formation of cancer, sterility being to a

certain extent a protection against it. Among the female cancer patients, 55 out of 315 were single; and among the uterine patients taken separately, there were 12 single women out of 135. Of the married women, 86 per cent. among the uterine patients, and 71 per cent. of those suffering from cancer in other organs, had borne children. The average number of children produced by each married woman, was 5·2 among the uterine cases, and 3·89 among those with cancer in other situations.

3. The duration of life was found to vary greatly, according to the seat of the disease. In the cases of cancer of the breast, those who had been operated on, lived fifty-three months, whilst those in whom the disease was allowed to take its natural course, lived only thirty-two months.

4. As regards the hereditary nature of the affection, it was found that cancer was traced in $8\frac{3}{4}$ per cent. of the cases. There were five instances in which the patients had two cancerous relations, and in one very remarkable case five relatives were affected with cancer.

5. Phthisis was traced in 37 per cent. of the families of the cancer patients.

6. In reference to secondary cancer, it was found that the disease was either local, or did not extend beyond the lymphatic glands in about half the cases. There were secondary tumours in 79 per cent. of the breast cancers, in 23 per cent. of the uterine cases, and in 54 per cent. of the instances of true cancer in other organs.

7. That a great variety of diseases was found in the bodies of those dying with cancer. Tubercle was found in 15 out of 172 examinations.

8. That cachexia appears to be rather an effect of ulceration than the sign of a state of system preceding the evolution of cancer.

In conclusion, my best thanks are due to all the medical officers of the Middlesex Hospital, for their constant kindness in affording me every opportunity of observing their

cases, and also for placing their own records at my disposal. My obligations are especially due to Messrs. Arnott, Shaw, De Morgan, Moore, and Henry, for their kind counsel and aid during the time I have been engaged in collecting the information which has here been analysed.

ON THE

FORMS AND STAGES

OF

BRIGHT'S DISEASE OF THE KIDNEYS,

WITH ESPECIAL REFERENCE TO DIAGNOSIS AND PROGNOSIS,

(FOURTH COMMUNICATION.)

BY

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It will probably be conceded by most persons who have given any consideration to the subject, that the question of the relationship which exists between the various conditions of the kidney which are commonly included under the term Bright's disease, is one not only of speculative interest, but of practical importance.

That the appearances of the kidney in different cases of this, so-called Bright's disease, are very diverse, is sufficiently obvious. In one case the kidney is found large, vascular, and congested; in another large, pale, anæmic, and wax-like; in a third also large, pale, and anæmic, but

having numerous fine, yellow granulations like specks of sand scattered through its cortical substance. Again in other cases, the kidneys are more or less reduced in size, and their surface is uneven and granular.

Now there are some pathologists who maintain that, different as are the appearances presented by the kidney in these cases, they are nevertheless the result of a single morbid process. Bright's disease, according to these authors, is ever one and the same disease; and the various morbid appearances in the kidney, to which it gives rise, result simply from the greater or less degree of intensity of the disease, and the more or less advanced stage at which the renal degeneration has arrived before the fatal termination.

That this theory of the *oneness* of Bright's disease is untenable, and inconsistent with the clinical history and the morbid anatomy of the kidney in its various conditions has been, I think, abundantly proved by myself and others who have written on the subject.

Amongst the opponents of the theory of the *oneness* of Bright's disease I may particularly mention Dr. Todd[1] and Mr. Simon;[2] Dr. Wilks, too, has ably advocated the doctrine of a diversity of the forms of Bright's disease;[3] and Dr. Richard Quain, in a paper published in the 'Lancet'[4] nearly fourteen years ago, argued for the recognition of three principal forms of the disease, namely, 1, the simple enlarged mottled kidney; 2, the truly granular or atrophied kidney; and 3, the large, flabby, fatty-looking kidney. Dr. Quain's remarks on this subject, published at a time when microscopic examinations of morbid kidneys were in their infancy, deserve more notice and credit than they have received.

The *chronic* forms of Bright's kidney, of which only it is my intention to speak in the present communication, may

[1] 'Clinical Lectures on certain Diseases of the Urinary Organs.'
[2] 'Med.-Chir. Transactions,' vol. xxx.
[3] 'Guy's Hospital Reports,' 1852-3, p. 232.
[4] 'Lancet,' November, 1854, p. 594.

conveniently be arranged in two main divisions; 1st, the large white kidney, whether simply pale, anæmic, and wax-like, or containing more or less oil; and 2d, the small, contracted, granular kidney.

The chief points of distinction between the large white Bright's kidney and the contracted kidney are these:

1st. The urine secreted by the large kidney is less abundant, of higher specific gravity, more constantly and copiously albuminous, and usually contains clear fibrinous wax-like casts, either with or without oil, but none of the granular casts which are thrown off from the tubes of the contracting kidney.

2d. The minute anatomy of the two kidneys is very different.

In that form of disease which leads to the contracted kidney, the gland-cells which line the uriniferous tubes undergo—even at the very commencement of the malady, and sometimes long before the urine contains a trace of albumen—a process of disintegration; as a result of which they become detached from the basement membrane, and appear as a sediment in the urine in the form of the granular tube-casts just now mentioned. The uriniferous tubes thus become entirely denuded, or they are found to be lined by a layer of small, transparent cells, entirely different from the normal glandular epithelium. It is probable that a tube thus denuded, has lost, in a great degree, its power of secreting the solids of the urine, while it may yet be able to separate the watery constituents from the blood. And hence, perhaps, the abundant flow of urine, pale in colour, and low in density, which characterises this form of disease.

In the case of the large white kidney, on the other hand, the gland-cells do not become disintegrated, detached and washed out, so as to leave the tubes denuded. They remain, for the most part, adherent to the basement membrane, and undergo changes, varying from a slight granular opacity to a complete oily degeneration, or they may at length become replaced by an albuminous or a fibrinous material, which

more or less fills and obstructs the tubes. This condition of the renal tubes appears to be less favorable for the transudation of water than the denuded condition before described.

Another very important distinction between the two forms of disease, is to be found in the relative frequency of *dropsy* as a symptom. There are very few exceptions to the rule, that patients dying with the large Bright's kidney have had dropsy, in a greater or less degree, at some period of their history. Whereas, the majority of those who die with a contracted kidney, have not suffered from dropsy in any form or degree. I have lately been engaged in analysing the main features of about 300 cases of Bright's disease which have come under my observation; and amongst other points for inquiry, I have endeavoured to ascertain the relative frequency of dropsy in cases of the large and the small Bright's kidney respectively. For this purpose it is necessary to exclude entirely from consideration all that numerous class of cases in which valvular disease of the heart or other acknowledged and sufficient cause of dropsy coexists with the renal disease.

I find, then, that out of twenty-six fatal cases of enlarged Bright's kidney, there was dropsy in twenty-four; the proportion being, in round numbers, 92 per cent. And in many of the cases the dropsy was great, general, and of long duration.

Whereas, of thirty-three fatal cases of contracted kidney, there had been dropsy in only fourteen, the proportion being 42 per cent.; and in most of these cases the dropsy was very slight and partial.

Surely these facts are fatal to the hypothesis, which assumes that a contracted kidney is, in every case, but an advanced stage of the same form of disease as that which gives rise to the large white kidney. If all the contracted Bright's kidneys have passed through a previous stage of enlargement, it is difficult to understand how it can happen that the majority of those patients who have reached this final stage of renal degeneration, should escape the dropsy,

which, in a greater or less degree, troubles nearly all those who die in what is assumed to be an earlier stage of the same disease.

It is, I think, sufficiently obvious that there is an intimate relation, on the one hand, between the comparatively scanty secretion of highly albuminous urine by the large Bright's kidney, and the frequent occurrence of dropsy in these cases; and, on the other hand, between the copious secretion of watery urine by the small kidney, and the comparative infrequency of dropsy as a symptom. The risk of dropsy being, *cæteris paribus*, directly as the drain of albumen from the blood, and inversely as the secretion of water by the kidney. In other words, the more scanty the secretion of urine, and the greater the amount of albumen which it contains, the greater is the probability that the patient will become dropsical.

Now, I maintain, as I have done for years past, that the small Bright's kidney, through which there usually flows a large daily measure of watery urine, is the result of a different series of morbid changes from that which gives rise to the large white kidney. The rule is that a large Bright's kidney remains large to the end, and does not become a small one; and, on the other hand, a contracted Bright's kidney does not pass through a previous stage of enlargement.

Having thus stated what I believe to be the rule with regard to this subject, I come to the chief object of this communication, which is to point out some exceptions to this rule, exceptions which, when carefully considered and rightly interpreted, tend, as I think, to prove and confirm the rule.

There are undoubtedly cases of Bright's disease in which the kidneys having become enlarged, subsequently undergo a process of contraction, in a greater or less degree. These cases, however, are so exceptional in many of their most important features, that they afford a remarkable confirmation of the doctrine that in ordinary cases the contracted Bright's kidney is not an advanced stage of a previously

enlarged kidney, but rather that it is the result of a distinct series of morbid changes.

The cases of Bright's disease in which contraction of the kidney has followed upon enlargement may, for convenience of description, be divided into three classes.

First, there are cases in which the size and weight of the kidney are found after death to be considerably increased, yet in the cortical portion of the gland there are unquestionable indications of commencing atrophy and contraction. The cortex of the kidney in these cases is anæmic, pale, and wax-like, the surface being more or less uneven and nodulated, while the thickness of the cortical substance appears diminished in a greater or less degree.

I have notes of six cases in which the kidney was found thus enlarged yet partially atrophied. The surface and section of the kidney in one of these cases (that of a man named Revels) is represented in a plate from an original drawing by Dr. Westmacott. I have no note of the weight of the kidney in that case, but in the other five cases it was as follows: In one the two kidneys weighed thirteen ounces. In one, a boy aged fifteen, their combined weight was ten ounces, and in another man they weighed together twelve and a quarter ounces. In one the weight of a single kidney was seven ounces, and in another eight ounces. Now, as in each of these cases, there was more or less atrophy of the cortical portion of the kidney, we have so many unquestionable examples of contraction following upon enlargement of the gland.

In a second class of cases this contraction of a white and waxy kidney had proceeded farther; so that the size and weight were reduced below the average of the healthy organ.

I find three cases of this kind in my note-books. In one case a single kidney weighed four ounces, in another the weight of the two kidneys was eight and three quarter ounces, in the third case the weight of the kidney was not noted, but the gland was of small size, and its surface coarsely granular.

The third class of cases are those in which the kidney, having become enlarged and undergone *fatty degeneration*, has subsequently contracted, the fat granulations being still visible in the atrophied gland.

I have notes of five cases of this kind, the weight of the two kidneys varying from seven ounces and three quarters to six ounces and a half.

The second and third of these classes of cases call for some additional remarks, and I will speak first of the atrophied granular fat kidney.

That a fat kidney is generally of large size will scarcely be denied by any one, and that the fatty disease has no great tendency to produce atrophy and contraction of the gland is conclusively shown by the fact that the fat kidney is usually found to be of large size after the disease is known to have existed for many months, and even for several years.

During the last fourteen years I have met with only five cases in which a *small* fat kidney was found on post-mortem examination. And during the same period I have seen only three cases besides, in which I inferred from the character of the urine, and especially from its microscopic appearances, that a fat kidney was becoming atrophied.

The diagnosis of these cases during life is sufficiently certain and easy. In two of the five cases above mentioned the commencement and the progress of the atrophic change in the were clearly ascertained by repeated examinations of the urine; and the casts and cells preserved as microscopic specimens at different stages of each of these cases retain their characteristic appearances at the present time; so that the most important signs marking the progress of a disease, which ended fatally more than two years ago, are still open to the inspection of the microscopic observer.

The following is an outline of the history of one of these cases:

Stephen G—[1] was first admitted into King's College Hospital in October, 1846; his age being then twenty-three. He was suffering from acute general dropsy with albuminous and bloody urine. The dropsy disappeared during his stay in the hospital, but the urine continued to be albuminous, and it deposited tube-casts and cells containing oil. In short, there was evidence that the kidney was undergoing fatty degeneration.

I saw him occasionally for several months after he left the hospital. His general health continued good, and he had no return of the dropsy, but the morbid characters of the urine remained unchanged. I then lost sight of him for several years, in fact until October, 1855—when he again came under my care at the hospital. He then stated that for the previous six years he had been serving as steward on board an Antwerp steamer, and had enjoyed excellent health until January of that year (1855), when he was suddenly seized with hemiplegia, from which he had but partially recovered; the leg being still very weak, and the arm nearly powerless. He thought that his paralytic attack might have been induced by his having for some time before taken daily a large quantity of Scotch whisky. Remembering his previous history, I was now much interested in ascertaining the condition of his urine. We found that it was still highly albuminous, and that it still deposited oily casts and cells.

Here, then, was a case of fat kidney of nine years' duration. But during the next few months, while the patient still remained under my observation, the deposit in the urine underwent a marked change. The oily casts and cells gradually diminished in number, and were replaced by the large granular and large waxy casts. I now felt sure that the secreting cells of the kidney were being rapidly destroyed, and that a process of atrophy had commenced; and I frequently spoke of the case as one in

[1] The early history of this case is fully given in the author's work on the 'Diseases of the Kidney,' p. 411.

which the somewhat rare transition from a large fat to a contracting kidney was in progress.

Fig. 1.

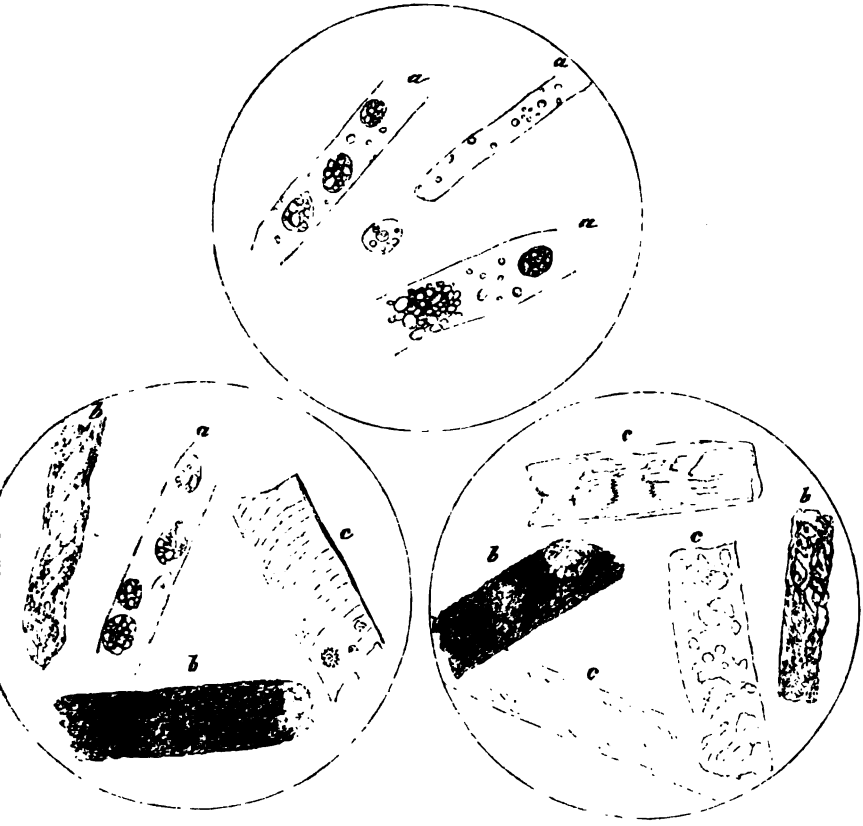


Fig. 2. Fig. 3.

Sediment in the urine at three successive periods of the case of Stephen Grey, × 200.

Fig. 1. Period of fatty enlargement.
„ 2. Commencing atrophy and contraction.
„ 3. Advanced atrophy and contraction.
a a a a. Oily casts.
b b b b. Granular casts.
c c c c. Large waxy casts.

The patient died in the hospital from uræmia, without any return of dropsy, in October, 1857, exactly ten years from the time of his first admission with acute renal disease.

The kidneys, the appearance of which is represented in Plate III, from Dr. Westmacott's drawing, were found much reduced in size, their combined weight being only seven ounces and three quarters; and the yellow fat granulations, which were still plainly visible, showed that contraction of the kidney had followed upon fatty degeneration of the glandular tissues.

The appearance presented by a contracted fat kidney, with its characteristic small yellow granulations, is so remarkably different from that of a small kidney which has been the subject of the chronic desquamative disease, that it is scarcely possible to mistake the one for the other, or, as I think, to doubt that they have resulted from a different series of morbid changes.

It is, however, not so easy to identify the second series of cases before referred to; namely, those in which a wax-like, but not a fat kidney, having once been enlarged, contracts until its size is less than that of the healthy organ. Yet it is not difficult to distinguish even these from the kidneys whose atrophy, having resulted from the crumbling and desquamation of their gland-cells, has not been preceded by a stage of enlargement.

The chief points of distinction between these two classes of cases are the following. A kidney which has been *enlarged* by chronic Bright's disease is always more or less anæmic, pale, and wax-like in its cortical portion; and this appearance of anæmia does not pass away if the kidney should afterwards contract. On the other hand, the small kidney which results from the chronic desquamative process is often found to be comparatively red and vascular, even in its extreme stage of atrophy and contraction.

The different appearances presented by the kidneys in these two classes of cases will be found illustrated in Plates IV and V.

The contracted waxy kidney is usually much firmer and harder than the other, and its surface is more uneven and nodulated, or coarsely granular. Again, on microscopical examination, the appearance of denuded tubes, which results from the destruction and removal of the gland-cells, is much less frequent in cases of contracted waxy kidney than in cases of the chronic desquamative disease. Lastly, it is probable that a waxy kidney which has once been much enlarged never contracts to the small size often attained by the essentially atrophic form of Bright's disease. Only three out of the nine contracted waxy kidneys which I have met with were reduced below the normal size, and the smallest of these weighed four ounces; whereas, kidneys which have been wasted by the chronic desquamative disease are not uncommonly reduced to half that weight; and in the last volume of the 'Transactions of the Pathological Society of London' (vol. ix, p. 308), Dr. Wilks has briefly reported the case of a woman whose kidneys were so extremely degenerated, that "the two together weighed less than one ounce and a half."

I think, then, that a careful observer would scarcely fail to distinguish between these two forms of disease, from an examination of the kidneys alone, and without any previous knowledge of the patient's history. But the distinction will be easier if the progress of the disease has been observed from the beginning. The early history of these cases of contracting waxy kidney is that of a chronic enlarged Bright's kidney. In some instances the disease originates in an attack of acute dropsy with albuminuria; in other cases its origin is latent, but dropsy in some form or degree usually occurs sooner or later during the progress of the disease. The urine, moderate in density and quantity, is copiously albuminous, and it is either free from sediment, or perhaps it deposits a cloud, which is found to contain the small, clear, wax-like casts. At a later period the sediment in the urine is dense and abundant, being in great part composed of the large granular and waxy casts, the diameter of which is equal to that of the

uriniferous tubes. These appearances in the urine mark the stage of atrophy and contraction of the kidney. The destruction of the renal epithelium by disintegration, and the choking of the tubes by unorganized fibrin, appear to be constantly associated with atrophy of the glandular tissues; and the rate of progress of the degeneration may be accurately estimated by the amount of the before-mentioned dense sediment in the urine.

It is a well-known fact that patients who have constantly a large amount of albumen in the urine, may go on for many months, and even for years, without any serious impairment of their general health. In cases of this kind the entire absence of sediment, or the presence of a light cloudy deposit containing a few small waxy casts, without oil, is the most favorable condition of urine; while an increase in the amount and density of the urinary sediment, and an abundant appearance of the large-sized casts, are signs of unfavorable import, indicating, as they do, a rapid destruction and displacement of the renal gland-cells, which in all probability will be soon followed by the alarming symptoms of suppressed secretion and uræmic poisoning.

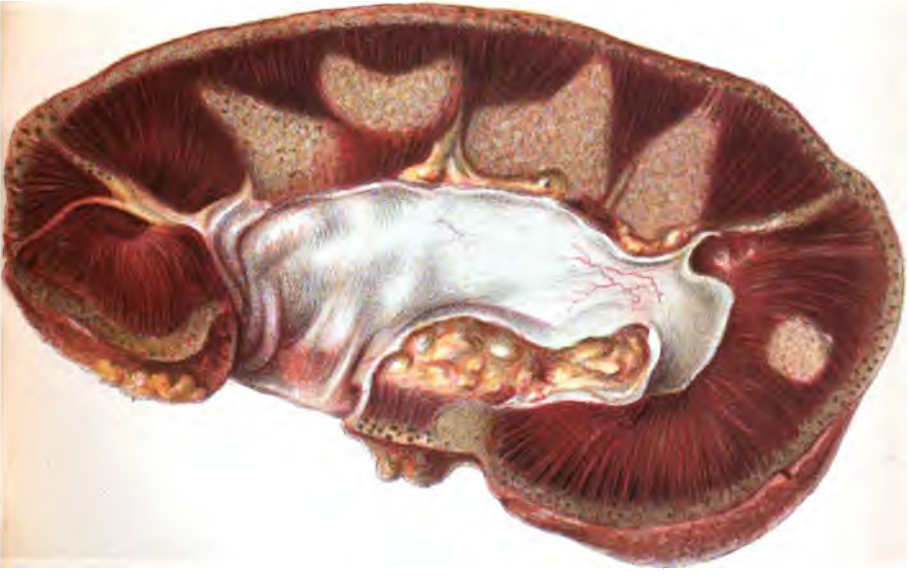
DESCRIPTION OF PLATES III, IV, AND V.

The illustrations are intended to represent the characteristic appearances of three different forms of contracted Bright's kidney.

Plate III.—The surface and section of the kidney in the case of Stephen G— (p. 160). The gland has been enlarged, and has undergone fatty degeneration; it has subsequently contracted to less than the natural size, the small, yellow, fat granulations being still visible in the anæmic and atrophied cortical substance.

Plate IV.—A large, waxy kidney in process of contraction. The entire gland is larger than natural, but the section shows the cortical substance much reduced in thickness, while the surface is uneven, and presents an irregular blending of wax-like anæmia with congestion. (pp. 161-2.)

Plate V.—An extremely contracted kidney, the result of the "chronic desquamative disease." The section shows that the cortical substance has nearly disappeared, yet the capsular surface of the kidney retains an appearance of redness and vascularity (pp. 161-2).



AN ACCOUNT OF THREE CASES

OF

ANEURISM OF, OR WITHIN, THE ORBIT,

TREATED BY LIGATURE OF THE COMMON CAROTID ARTERY;

WITH OBSERVATIONS.

TO WHICH IS ADDED A REPORT OF A FOURTH CASE, TREATED SINCE THE PAPER WAS READ.

BY

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ANEURISM by anastomosis, as it is called, of the orbit, is a disease of comparatively rare occurrence. Indeed, judging from works on general Surgery, as well as those devoted exclusively to ophthalmic practice, it would appear that few of the writers had met with a case; for in many of the works the affection is hardly, if at all, mentioned; and where it is spoken of more fully, reference is made, not to cases treated or seen by the writers themselves, but to five cases recorded in the 'Transactions' of this Society, and some three or four others. For a great number of years, the cases of Mr. Travers, in the second volume, and that of Mr. Dalrymple, in the sixth volume of the 'Transactions,' stood alone, so far as my information extends. In the twenty-second volume, Mr. Busk published an interesting case, following a violent blow on the head, with concussion

of the brain, which had fallen under his care, and also shortly referred to another, treated by Mr. Scott; and in the thirty-seventh volume, Mr. Curling has given an account of a very similar case of traumatic aneurism within the orbit, which he treated in the London Hospital in the year 1854; and he again refers to Mr. Scott's case, which he had seen.

Mr. H. Walton has recorded a case of aneurism by anastomosis in the orbit of an infant;[1] and, through the politeness of Mr. Erichsen, I learn that, in the year 1859, he saw Velpeau, in Paris, tie the carotid in a similar case; and also that Jobert about the same time did so in another. These are, I believe, the two cases recorded in the 'Bibliothèque du Médecin Praticien,' as referred to by Mr. Walton (page 264).[2] Lastly, in an essay in the 'New York Journal of Medicine' for July, 1857, by Dr. J. R. Wood, of that city, entitled, "The Early History of the Operation of Ligature of the Primitive Carotid Artery,"—where a very large number of cases of ligature of this vessel for various affections, by himself and others, particularly Dr. Valentine Mott, are mentioned,—I find there are three which may now be alluded to, though two of these cases can hardly, I think, in strictness, be included in the limited list of cases of aneurism of the orbit. One was that of an infant, born with a small nævus, which by the sixth month had so involved the right cheek and orbit, and, having ulcerated, had given rise to the loss of so much blood, that Dr. Wood tied the carotid. This operation had the effect of checking the hæmorrhage, though three years afterwards, when the child was lost sight of, the tumour of the integuments still remained large. In a second case, aneurism by anastomosis in the orbit had passed over the

[1] 'Treatise on Operative Ophthalmic Surgery,' p. 259.

[2] Mr. Walton does not mention the volume of the 'Bibliothèque du Médecin Praticien' in which the report of these two cases is to be found. Unfortunately I am not able to supply the omission, having searched the libraries in London in vain for the periodical, which, I believe, has been discontinued for several years past.

bridge of the nose into the other orbit. At a very early age Dr. Mott tied the carotid with success. The third was the case of a young man with symptoms of fracture of the base of the skull, occasioned by the walls of a house falling upon him: there was exophthalmia, with aneurismal thrill, and Dr. Van Buren tied the left carotid with success. This third case of traumatic aneurism, in its main features, strongly resembles those of Mr. Busk and Mr. Curling, just referred to. Mr. Guthrie, in his work on the 'Operative Surgery of the Eye,' shortly mentions that, in the post-mortem examination of the body of a man, true aneurism of both ophthalmic arteries was found. Other cases, as one by Dupuytren, a second by Schmidt, and a third by Freer, all of doubtful nature—the last being probably fungoid disease,[1]—are also published; but, so far as I have been able to ascertain, the above are all the undoubted cases which have been recorded. As it has happened to me to have had the treatment of three cases, in all of which I have tied the carotid artery, I have thought it proper they should be recorded; and, considering that five out of the very few known cases have been given to the profession through the 'Transactions' of this Society, that no other medium is so proper as this for their publication. It will be better to give a short narration of the cases first, as each presented some points of interest peculiar to itself, and as one of them—fortunately I was almost about to say—ending fatally, enabled me to examine the

[1] 'Observations on Aneurism and some Diseases of the Arterial System,' by George Freer; who not only calls the disease fungus hæmatoides, but gives a coloured plate of the patient, which appears to me to represent as accurately as possible a large fungoid mass sprouting from the orbit. Yet Mr. Middlemore, who, if I rightly understand his statement, saw this case while it was under Mr. Freer's care, says it was not fungoid disease, but aneurism by anastomosis, as "is quite evident from the history of its progress, the age of the patient, combined with the throbbing pain he experienced in the orbit, the ramification of vessels upon the surface of the tumour, and the great amount of hæmorrhage which took place from them." (Middlemore on 'Diseases of the Eye,' vol. ii, p. 619.)

parts after death. I will then make such observations as appear proper.

The second case may be shortly related, but the first and third require to be given more in detail.

Case 1.—S. W—, an active, well-made, healthy man, applied to me on account of the condition of his left eye. He was then at his work, and so continued until the end of October, three days before the operation. His history of the affection was reserved and inconsistent, it was only at intervals and in parts, and not until long after the conclusion of the case, that I could learn all the particulars. As he was an intelligent man, I could not understand this at the time; but it was explained by his subsequent career. He was an overlooker in a flax-mill, amongst delicate and rapidly moving machinery, where great attention and good sight are requisite. He was regarded as a confidential servant, a character which he did not merit; for, though never away from his work during the day, his nights were spent in poaching or worse pursuits. Hence his object to conceal the origin and progress of the affection, and hence also greater difficulty in the diagnosis and treatment.

It would appear that he had been at a shooting-match, got drunk, fought, and received a blow upon the eye, by which the lids were much swollen and discoloured. This passed off, and, as he said, he felt no inconvenience; he regarded it as an ordinary black eye, and quite forgot it when I spoke to him on the subject. However, after a few weeks, he perceived that the eyeball looked and felt different from the other, that it was bloodshot; the sight was dim; there was difficulty in opening the lids on getting up in the morning, and there was some aching about the orbit.

At this period (August 14th, 1852) when I first saw him, the globe was decidedly prominent; the conjunctiva much congested, its vessels being large rather than very numerous; the movements of the ball were a little im-

peded; the iris was natural in appearance, but somewhat sluggish in its motions; the sight was not materially affected. He did not complain of much pain, but a feeling of distension annoyed him.

I thought the globe was pressed forwards by some effusion posterior to it, either a chronic abscess or a serous cyst. Mild continued irritants behind the ear, a lead wash to the eye, mercury at bedtime, with bitter aperients in the morning, and a total abstinence from stimulants, were prescribed. I saw him only occasionally. At first he thought he decidedly improved, which, however, I did not; and probably he only thought so because his general health became better. Gradually the ball became more prominent; the congestion apparent in the sclerotic, as well as in the conjunctiva; the iris dull and more sluggish; the lids much swollen; the upper one, in its middle part, bulged considerably; the lower one at both its inner and outer angles, rather as though from some deep-seated fluid than from congested palpebral vessels. Pressure on either of these projections rendered the others more tense, and it appeared as though the skin would shortly give way from distension.

On October 26th, the outer prominence of the lower lid was punctured with a broad needle. Only a drop of arterial blood followed. Thinking there might be a cyst which had not been opened, I punctured the projection in the middle of the upper lid, when a minute jet of arterial blood spirted out, but it ceased on the withdrawal of the needle. The conjunctiva at once became intensely chemosed, and the lids greatly distended from the infiltration of blood into their tissues. By cold water it was at once checked. I now felt sure that an aneurism of the orbit existed, of which I had before some suspicion. Moderate pressure over the orbit with lint dipped in cold water, and active purgatives, were directed.

October 28th.—The distension and discoloration of the eyelids are less, but I am now satisfied there is some pulsation in the orbit. He for the first time informed me

that, for some weeks after the blow, he had been under medical advice for excessive pain on the left side of the face and head, which had been considered and treated as tic douloureux. He now also said that, ever since the blow, at night he had been much annoyed with a constant beating noise in the head and a singing in the left ear, but that, except on stooping, until the last two days, he had not felt it during the daytime. Probably the being all day until now amongst the noise and vibration of the machinery may explain this. To continue purgatives, pressure, and cold.

November 3d.—The distension of the lids has so far subsided that they can be opened with the fingers sufficiently to expose the ball. This has become more prominent than it was a week ago, the congestion of the conjunctiva is greater, the pulsation has become much more evident, and there is a decided thrill. As pressure upon the carotid at once stops these symptoms, and the eyeball becomes less prominent and paler, it has been determined to tie the vessel. There was no difficulty in the operation. The sheath of the vessel was only opened just to admit the needle. Neither the vagus nerve nor jugular vein was seen. At the moment of tightening the ligature he felt pain and nausea; both of which, however, as instantly passed off, and he exclaimed, "The pain and noise in my head have gone, and I can see better;" and he became, on losing the noise and confusion, more conscious of having had them for some time past. The eyeball at once became paler and less prominent. The pulse was unaffected, or, if anything, it was rather slower and more feeble.

5th.—He has gone on well until to-day. He now complains of headache, particularly on the right side; is easy if lying on the left side, but if he turns to the right, there is great pain on that side of the head, but none on the left side. Towards night this was increased by the noise of cannon and fireworks, which he complained of as having gone painfully through the right side, but not affecting the left. Bled from the arm, with relief.

12th.—So well that he will get up. The head is quite comfortable. He can open the eyelids; the ball is receding into the orbit; the congestion of the sclerotic and conjunctiva is nearly gone, except on the outer side of the ball.

27th.—Up to three days ago he had progressed well, the eye becoming nearly natural, and the lids being well opened. Since then he has not improved, and I think there is a little return of pulsation in the left temporal artery, with a little more fulness in the eyelids. To be bled to sixteen ounces, and well purged.

December 4th.—Condition of eye not much altered. Pulsation in temporal branches gone. Says he is so well that he will go home. As the ligature is quite fast, and some fungus is sprouting up by its side, it has been cut off within the cutis. Except in the course of the thread the wound immediately healed.

January 23d, 1859.—As I found that, on going home, he at once went out poaching during the night and rabbit-shooting during the day, he was directed to go to his work, at which he has continued since the last report. He considers himself quite well, and takes as much exercise as ever. The condition of the eye has gradually improved in every respect. During this last night great pain has come on in the left eye, the ball of which is much more projecting; the congestion of the conjunctiva has much increased, and I fancy that I can detect a feeble pulsation, though not with certainty, in the orbit. He has been constantly purged, had cold water to the eye, and declares that he has been quiet at night and abstained from all stimulants (a statement which he afterwards confessed to be false, and that he had been out all the night poaching). Though the pulse was, and had been rather weak and slow, he was bled from the arm to twenty-four ounces, ordered to take as little food and drink as possible, to remain in an upright position, and to continue the cold and pressure.

28th.—The pulsation and bruit have decidedly increased during the last week, as have all the symptoms; so much so that I have determined to tie the right carotid (which

the man is wishful to have done at once) if the disease be not lessened within a week. Venesection repeated.

31st.—All the symptoms are much improved. He states that, up to going to bed last night, he was not at all better; but, on waking this morning, he found the improvement had taken place during sleep.

February 5th.—In the same condition. Venesection repeated.

8th.—Has improved since the 5th; bled again to twenty-four ounces, without any indication of faintness. Last night the ligature came away, *ninety-six days after the operation*. For two or three days the cicatrix had been a little tender; the knot is well made and firm, the loop perfect, and the thread unaffected.

21st.—The improvement has slowly progressed; the pulsation and bruit are less, but not gone. As snow has fallen, I have directed the orbit to be covered with some, and for it to be constantly renewed as it melts.

23d.—For the last forty-eight hours snow has been continually applied. At first the aching induced was severe, but not after a time. The congestion and prominence are less; in other respects he is in the same condition.

This man now suddenly ceased his attendance. I subsequently learnt that he returned to his work for a few days, when he was committed to prison. On his release he left the town, and I lost sight of him until June 21st, when he presented himself. Since the last report he has done nothing, except the frequent application of house-leek and cream, which has caused great lachrymation. He has lived freely, and worked hard. He is in perfect health: the condition of the eye is much the same as it was a month after the carotid had been tied, and before the recommencement of the symptoms. The lids open nearly as wide as those of the other eye, though there is still some induration in them. There is little, if any, prominence of the ball. There are still the three large convoluted vessels

on the outer side of the conjunctiva, but no general congestion of it. He can see objects, but their outlines are indistinct. The iris is dull and sluggish. No pulsation in branches of left carotid; not more than normal in those of the right.

He was now again committed to prison; and on his release, worked as an excavator in making sewers. I saw him during the summer of 1857. The eye was then natural, except cataractous; there was neither congestion nor projection. He was now again convicted of housebreaking, and was sentenced to four years' penal servitude in Wakefield Prison, where I have seen him (March 8th, 1859). He is well, and doing ordinary prison-work. The eyelids are normal; the ball a little prominent; the sight is quite gone; there are two or three large convoluted vessels as before, but no general vascularity. He now says that he was perfectly cured by the operation, and has only himself to blame for the slight recurrence of the symptoms three weeks after the operation, when he clandestinely left the house on a very cold day, and ate and drank freely; and, also, that the greater recurrence, ten weeks afterwards, was caused by his being out, all night, poaching—facts which he at the time concealed. Even now, so far as the aneurism is concerned, the cure may be considered complete.

Case 2 is that of Thomas I—, æt. 38, wool-comber, a pale, feeble-looking man, as many wool-combers are. He, however, says that he has always been well and capable of work. He never had any injury of the part, and cannot in any way account for the disease. About thirty-four weeks before my seeing him, he noticed a small swollen spot in the lower lid of the left eye, with a difficulty of opening the lids. As the swelling enlarged, he applied for advice, which he pursued for twenty-eight weeks, during which time the disease steadily increased.

I first saw him in March, 1856, at which time both lids, but particularly the lower, of the left eye, were so much

swollen that he could not open them. When they were separated, the conjunctiva was seen to be much distended with serous effusion, and greatly congested. The eyeball was somewhat protuberant, and pushed upwards. The blood in the convoluted vessels was dark, more like venous congestion than active arterial enlargement. The lids felt boggy; there was little or no pulsation. By pressure on the part the ball could be made to recede, and the vessels emptied, but they soon filled again. The sight was very dim. Though he did not complain of the distressing pain and noise in the head of which the other two persons did, yet there was some confusion and bewilderment, sufficient, with the inability to raise the lids, to prevent his working.

Finding that by pressure on the carotid the congestion of the eyelids was materially lessened, and if the affected vessels were emptied by steady pressure over the lids and orbit, so long as the pressure was kept upon the carotid, the congestion did not return, and the eyelids could be kept partially opened (though I thought the veins were implicated rather than the arterial branches), and as he was extremely anxious to be cured, I determined to tie the carotid. This was done on March 8th. There was no difficulty in the operation; neither the vagus nerve nor jugular vein was seen. There was not the least effect upon the brain, nor any unpleasant symptom when the ligature was tightened. For a few days (like Case 1), he complained of some sore throat and difficulty of swallowing, with expectoration of bloody mucus. The swelling of the eyelids and congestion of the conjunctiva at once materially diminished. The wound healed without the formation of any matter, except a few drops in the course of the ligature, which did not come away for upwards of a month, when he was discharged. The ball had then returned to its normal situation; the congestion and serous chemosis of the conjunctiva had disappeared; the lids were much less swollen, and could be kept opened, though they, but particularly the lower one, continued somewhat congested and boggy. He soon resumed his work; shortly after which, as he was

wheeling a heavily laden barrow up a hill, he felt his eye suddenly become worse, the lids more swollen, and the sight impaired. He thought the disease was returning, but, by keeping quiet for a few days, he became better, and able to work again. I saw him in March, 1857, twelve months after the operation; at which time my memorandum is, "I— says that he is as well as he ever was in his life. He has continued constantly at his work as a wool-comber. The sight of the left eye is as good as that of the right—indeed, perfect. Both left eyelids are rather boggy, and somewhat enlarged, but the congested condition of the vessels is gone, and he does not experience any inconvenience whatever. There is no pulsation in the left carotid trunk, but in its branches there is very little difference from those of the other side, which pulsate feebly. The health is perfectly good."

Case 3.—On March 31st, 1858, I was consulted by Elizabeth H—, of Garforth, near Leeds. She is a widow, æt. 65, mother of fifteen children. She is a short, stout, thick-set woman, who has worked and fared hard. She says her health has been good, excepting two attacks of erysipelas of the head and face. About ten years ago she had some inflammation of the eyes, for which she had a lotion from the village druggist. In two or three days they were well. She has never had any hurt or accident. There is an oblique depression in the soft parts, and in the bones where superficial, extending from the middle of the left parietal bone, along the frontal, across the eyebrow and cheek, through the upper lip, which is thin and almost fissured in the part. This mark is congenital, and has never occasioned any inconvenience whatever. Her mother attributed it to having, while pregnant with her, fallen against the edge of a table, and cut her forehead badly.

Six weeks ago she was perfectly well. On February 17th she walked to Colton, a village between two and three miles from her residence, and, on returning home in the

evening, as she stooped down to take off her shoes, she suddenly, "as the crack of a gun," to use her own expression, felt something give way in the left eye. There was instantly great pain and buzzing noise in the head; she became confused and deaf on that side; the eyeball protruded, and felt as though it would burst, and was very red and painful; the lids, also, became swollen, and nearly closed. She had a bad night from pain. The next morning a medical man saw her, and directed the application of flour, as he thought another attack of erysipelas was coming on. There being no change in a few days, another medical man was consulted, who told her there was a tumour of the eye, for which he could do nothing.

At present the left eyelids are quite closed, much distended, and greatly congested, with large superficial veins. When the lids are separated, the eyeball is seen to be considerably protruded; the iris is motionless; the lens muddy; the conjunctiva greatly chemosed and scarlet, with several large convoluted dark veins. The sight is gone. There is a most distinct bruit, and a decided pulsation, synchronous with the pulse. Both these are particularly evident on the inner side of the orbit. There is a perpetual beating and buzzing noise in the head, from which she feels confused and bewildered. On stooping or lying down these are increased, and she feels as though the eye would burst. From the continual pain and noise there has been want of sleep, to which she ascribes at present feeling very unwell.

There being apparently no doubt as to the nature of the disease and the propriety of tying the carotid, I explained this to her and her friends, directing, while she decided and made the necessary arrangements for leaving home, the use of a cold wash and sedative salines.

The operation being decided upon, it was performed on April 3d. Unlike the two preceding cases, it presented considerable difficulty, from the following causes.

The neck was short, thick, and fat. There was a large bronchocele, reaching nearly to the sternum, and overlap-

ping the carotid sheath. The veins of the neck were very large, full, and numerous. The sterno-cleido-mastoideus muscle was very broad, and the common carotid divided unusually low down. When the sheath was exposed at the upper edge of the omo-hyoideus muscle, the vessel was seen dividing into its two branches, so that the wound had to be enlarged to give room for the ligature to be carried below this muscle. The sheath was here barely punctured, to allow the needle to pass in on the inner side of the artery. Neither jugular vein nor vagus nerve was exposed. Just as the ligature was carried round the vessel, and the eye of the needle brought up, there was a sudden gush of florid blood in a considerable stream from behind the sheath. The vessel from whence it came could not be seen. On tightening the ligature the bleeding instantly ceased. There was some troublesome bleeding from two or three divided veins, which, however, stopped in a few minutes. After a quarter of an hour the wound was closed by elastic (caoutchouc) sutures and plaisters.

All pulsation and bruit in the orbit at once ceased. The eyeball receded, and the conjunctiva became blanched. The noise and distress in the head (as she subsequently said) disappeared. But, instantly on the tightening of the ligature, there was some convulsion of the *left* side, while the *right* was motionless. There was partial unconsciousness, with sighing, and an appearance of nausea and faintness, evidently more from the condition of the brain than of the heart, for the pulse remained nearly natural. A little ammonia soon rallied her, but for a few minutes appearances were alarming, and it was doubtful if she would rally. For several hours there was only partial consciousness, with some difficulty of speaking. The faintness much resembled that induced by tobacco in those unaccustomed to it. Reaction soon came on, and the branches of the right carotid pulsated vigorously.

4th.—She has passed a good night, and is now nearly herself. The head is clear, and free from pain and noise; the pulse is good and regular, not more than 70. There

is great difficulty in swallowing, and some in talking. The throat is sore. Vessels on right side of the head full, those on the left not to be felt. Left eye quite level with the right one; conjunctiva pale; sight improved.

5th.—In the morning she was in a satisfactory state; but towards evening she became very restless, with incessant motion of the left side of the body. Bowels not having been opened, a purgative was given.

6th, a.m.—Passed a bad night, is dull, very restless, and wandering; has great difficulty in speaking, so as to be hardly understood, which appears to depend more upon the brain than the tongue, as she says some words well enough. She has been freely purged. The pulse is weak and not quick. To have good support and ammonia, with a sedative.

6th, p.m.—Is decidedly better in every respect; is now conscious, speaks better, swallows well, and says the throat is not now sore. The pulse is natural. Wound nearly healed.

7th.—Has quite lost the use of the right side. She lies with the eyes shut, as though asleep, but on being spoken to, she hears and answers sensibly, articulating some words distinctly, but most as though only muttering. When told to put out the tongue, she makes no effort to do so, though she is continually putting it out to lick the lips. The respiration and pulse are natural. She has taken sufficient food without any difficulty. The eye is natural.

10th.—Improved in every respect. Last evening, while asleep, her nurse left her. On returning, she found her with the head on the floor and the feet upon the bed, a very high one; she had either fallen, or attempted to get out of bed. It does not appear to have affected the wound. She speaks better, is more herself, and has a little power in the right hand.

11th.—Improvement continues. The left eyeball is quite natural, and the sight of it good.

12th.—Called up at four o'clock this morning, owing to hæmorrhage from wound. There were only a few drops of blood—not sufficient to detach the plaister—and it had quite ceased when I got to her; she had been rubbing the part.

When the wound was dressed to-day, there was found some acrid discharge along the ligature, which had excoriated the skin, and which probably had caused her to meddle with the wound. The paralysis has improved, but not the restlessness; indeed, with more power she moves more, constantly rolling over; and when told to lie still, she is exceedingly cross and spiteful.

13th.—Called up again at four o'clock this morning. There had been a sudden gush, and at least half a pint of blood lost; it had, however, entirely ceased when I got to her. There was no return during the day, and she rallied considerably. While under the influence of an opiate she was quiet and slept; otherwise she was in constant motion, rolling over from side to side, violently and suddenly raising the head and stretching out the neck, pulling and rubbing the end of the nose, as though it itched excessively, but she either could not, or would not say why she did it. She could put out the tongue well; some words she could articulate distinctly, others not.

16th.—During the last forty-eight hours hæmorrhage has recurred six times, but has always been arrested by cold water, though once nearly a pint of blood was lost. The ligature is quite fast, and the wound is everywhere healed, except in its course, from whence there is some acrid discharge; there is also some swelling, as though matter were forming about the sheath of the vessel. A daughter, who came to-day, told me for the first time that the woman had been in the habit of taking opium.[1] To have a full opiate at bedtime.

17th.—In much the same condition; bleeding has twice recurred, but not to any great extent. The mental condition more resembles insanity than anything else. She appears to understand what is said to her, but to be incapable

[1] The account of opium-taking was most contradictory, one daughter saying she took it, another that she did not. However, from subsequent inquiries I believe there is little doubt that she had taken it to a large extent since the occurrence of the disease, if not before; the pain of it being the excuse.

of replying, which, with inability to move the right side, renders her very irritable. She is constantly drawing attention to her right arm, by taking hold of it and lifting it up with the left hand.

19th.—The bleeding has recurred more than once, but never to any great extent, and always at once arrested by the application of cold water. There has never been bleeding when the wound has been dressed. This looked better, and the pus more healthy. At times she appeared to be rallying, but during the day she sank—sixteen days after the operation.

Examination of parts, forty-eight hours after death.—I had the greatest difficulty to obtain an examination of the body, and then only with a near relative standing by.

External appearance of both eyes exactly alike and natural. Brain very pale in colour, with unusually little blood in its substance or larger vessels; but, as far as could be judged, the quantity was alike on both sides of cerebrum and cerebellum. Blood thin, and of a red-pink colour. Arachnoid membrane everywhere thickened, and adherent to the cerebral surface, from old changes; there was no indication of any recent affection, nor any effusion. Both carotid and vertebral arteries everywhere, even in the smaller branches, patulous, and studded with earthy deposits. The choroid plexuses bloodless, white, and soft. Right hemisphere of brain firm and natural; whole of left decidedly soft, and in the lower part of the middle lobe, just by the side of the sella turcica, and above the entrance of the carotid artery, there was a patch of the size of a large hazel nut, quite soft and broken down. Here the left carotid, on emerging from the bony canal at the origin of the ophthalmic artery, was decidedly enlarged, and filled with, and surrounded by, a nodule of coagulum of the size of a large horse-bean. The right carotid was normal.

The contents of the left orbit, with the diseased curve of the carotid artery, were, as far as practicable under the circumstances, removed. The ophthalmic artery was considerably dilated, its coats thickened with atheromatous patches; two of its branches, particularly the inner, or the con-

tinuation of the trunk towards the inner angle of the orbit, were distended and filled with coagulum. The outer or lachrymal branch was also large, and filled with coagulated blood, but not to the same extent as the inner. (It will be recollected, before the operation it was at the inner side that the bruit and pulsation were most decided.) All the other branches, both of arteries and veins, were so small as hardly to be observed.

On removing the strips of plaister from the neck, the ligature was detached adhering to them; the knot was firm and securely made. The wound was perfectly healed, except at this spot, where the parts looked dark and somewhat sloughy. The thyroid gland was found very much enlarged, the middle lobe as a distinct projection; each lateral lobe reached down to the sternum. The ligature had been placed just below the bifurcation of the artery; the superior thyroid branch was also given off just at the commencement of the external carotid. Here ulceration had taken place, without the least indication of adhesion or coagulation having occurred on the upper side of the ligature; above it all the branches, as well as the two trunks, were quite open. Below, from the ligature to the sternum, the common carotid was well filled with an adhering coagulum. There was a large amount of fibrin along and within the sheath, consolidating all the parts together, except about the divided ends of the vessel, where there was not any. The jugular vein and vagus nerve were quite natural. The branches of the superior thyroid artery were all large and patulous.

The instantaneous appearance of the disease, the decided bruit and pulsation in the orbit, the distressing pain in the eye, the tormenting noise in the head, the loss of hearing in the left ear, the sudden cessation of all these on the tying the carotid artery, the serious effect of the operation on the brain, the convulsion of the left side of the body, the paralysis of the right, the mental alteration, the difficulty of speech and deglutition, the improvement in all of these, the falling off again in speech, the frequent hæmor-

rhage, the paleness of the brain and choroid plexuses, the evidence of old affection of the arachnoid, the atheromatous patches in the cerebral vessels, the continuation of this condition into the orbit in the two dilated branches of the ophthalmic artery, the enlargement of, and clot about the curve of the carotid near the origin of the ophthalmic artery, the softening of the brain exactly over the diseased artery, the large bronchocele, the low division of the common carotid into its two branches, the cardiac portion of the carotid trunk being filled with coagulum and consolidated with fibrin, while the distal portion was free from both, the congenital marking of the bones and integuments of the cranium and face,—all concur in rendering this an interesting and important case.

Have the cases, which from time to time have been recorded as aneurism by anastomosis of the orbit, really been of this nature? I believe not. Mr. Busk, in his comments upon the case in vol. xxii of the 'Transactions,' clearly showed that in those supervening upon direct violence to the head, the injury must be of the same character as traumatic aneurism in any other part of the body; in which opinion he was supported by Mr. Curling, in the report of his case in vol. xxxvii. Yet so strongly has the idea of Mr. Travers, of their being instances of aneurism by anastomosis, been impressed upon the professional mind, that the conclusive reasoning of these surgeons, and the almost self-evident character of the affection from its mode of origin, seems to have been overlooked; and in well-esteemed works on Surgery, published since these cases (at least since that of Mr. Busk) were brought under the notice of this Society, the old diagnosis is still adopted, and the affection is described as aneurism by anastomosis of the orbit, as by Mr. Fergusson, Mr. Erichsen, Professor Miller, Mr. H. Walton, and others. Even Mr. Guthrie,[1] who refers to the post-mortem examination of a case, in which during life

[1] 'Operative Surgery of the Eye,' p. 168.

the symptoms were very similar to those detailed by Travers and Dalrymple, by whom, however, "true aneurism of the ophthalmic artery of both sides" was found, still evidently adopts the idea of their cases having been anastomotic. Yet on reading Mr. Travers's description of his case (vol. ii), it is difficult to understand why he should have arrived at the opinion of it being aneurism by anastomosis, except that just before this time men's minds had forcibly been directed by John Bell to this condition of the blood-vessels, and that no such large branch exists in the orbit as is usually the seat of true aneurism in the limbs.

Equally so is it on perusing Mr. Dalrymple's account of the origin and progress of his case, except that of passive acquiescence in the diagnosis, which had emanated from so good a surgeon as Mr. Travers. Had the case witnessed by Mr. Travers been one of traumatic, instead of spontaneous origin, it is possible the world would never have heard of the definition so long current.

In hardly one particular do these cases of disease in the orbit resemble aneurism by dilatation or enlargement of the small blood-vessels in any other part of the body. 1st. It is very doubtful if aneurism by anastomosis is ever developed, unless it has had a congenital origin. Though occasionally its development into active disease is delayed until adult life, certainly, in by far the greater proportion of cases, it is so early in childhood. 2d. Aneurism by anastomosis does not appear suddenly; and when it is noticed, its increase is usually slow and gradual. 3d. It is not caused by direct violence. 4th. All the blood-vessels in the neighbourhood of aneurism by anastomosis appear to participate more or less in the increased action, as active agents, and not merely as passively dilated tubes. 5th. It is almost always, if not invariably, connected with the cutaneous or subcutaneous tissues. 6th. The result, where a single large distant artery has been tied in aneurism by anastomosis, is not such as to lead to the supposition that all pulsation and tumefaction would instantly disappear on ligature of the carotid, if such a disease existed in the orbit; though a cure might follow, the effect would be more gradual.

In contrast with these statements we have—1st. In aneurism by anastomosis, with the exception of Mr. H. Walton's case, where swelling was noticed before the infant was a month old, and pulsation when only four months; and the two congenital cases of Dr. Wood and Dr. Mott, where the integuments about the orbit were so involved that the affection should rather be regarded as that so frequently found in the integuments of the head and face, accidentally, from contiguity, invading the orbit, than as aneurism of the orbit itself; every other has occurred in adults. In my third case the woman was sixty-five years old. 2d. In no instance has there been any evidence of congenital undeveloped disease of the blood-vessels. 3d. In five of the cases the disease was the result of direct violence, as in Mr. Busk's, Mr. Scott's, Dr. Van Buren's, Mr. Curling's, and my first case; and in three others, to use the patients' own expressions, it came on "with a sudden snap on the left side of the forehead," "the attack was sudden, instantaneous," "as the cracking of a whip, so that she awoke in great alarm and leapt out of bed;" "suddenly, as the crack of a gun, I felt something give way" in the orbit, as in Mr. Travers's, Mr. Dalrymple's, and my third case. In one case only, my second, was the origin and progress of the attack unmarked by sudden or painful accession, and even in that the development was not slow. 4th. There has not been noticed any active, or indeed any participating enlargement in the neighbouring blood-vessels, the dilatation of the palpebral and conjunctival vessels evidently being passive congestion from posterior pressure. 5th. The disease has been deeply seated in the orbit, or even posterior to it, far away from the cutaneous structure, in every case, except the three infants of Dr. Wood, Dr. Mott, and Mr. H. Walton, where the disease was congenital. 6th. The tying the main artery leading to the disease has, in every instance in which it has been practised, as in true aneurism, at once arrested the pulsation, thrill, and tumefaction.

These reasons would of themselves appear to be conclusive against the idea of the disease being aneurism by anastomosis of the intra-orbital vessels, and sufficient to prove that it

must be either true, or circumscribed false aneurism; but the condition of the vessels found on post-mortem examination of my third case, and the aneurismal condition of the ophthalmic arteries in Mr. Guthrie's case, I apprehend, must be held as demonstrative upon the question, that not only are those which have a traumatic origin, but also those which occur spontaneously, not cases of aneurism by anastomosis.

It would appear that at least some, if not the majority, of the cases are false circumscribed or diffused aneurisms, resulting from rupture of the vessel rather than from dilatation of its diseased coats; and further, it would appear as not improbable that, while in some cases the disease may be in the ophthalmic artery itself, as in Mr. Guthrie's, in other cases it is within the cranium at the curve of the carotid artery, the ophthalmic branch being only secondarily involved, as in my third case, and possibly also in my second, where there was but little pulsation, and where the enlarged orbital vessels had the appearance of passively congested veins, as though there were some obstruction to the return of the blood into the cavernous sinus.

The first case is interesting, as showing how long a time may elapse from the receipt of the force occasioning the affection to the full development of it, and the comparatively small force applied directly to the eye, a blow with the fist, which occasioned the mischief. In the other four cases of traumatic origin—Mr. Busk's, Mr. Scott's, Dr. Van Buren's, and Mr. Curling's—the injury to the head was so great as to be the only apparent danger at first, the condition of the eye not at first appearing as abnormal. My first case is perhaps the longest upon record, in which a ligature remained upon so large a vessel as the common carotid, the patient during much of the time taking violent and continued exercise; for the man was certainly out poaching during many nights, and at his work during the day. Nor should the great increase in all the symptoms of the disease, after they had in a great degree subsided, and the disappearance of them under the application of cold to the part and diminution of the force of the circulation by repeated venesection, purging, and low diet, be overlooked; as it would induce a trial of these means, when from any

cause it were thought unadvisable to tie the carotid, or, having done so without success, to put them in practice before placing a ligature upon the other carotid; more especially as it appears that the woman, upon whom Mr. Travers operated, was nearly at a standstill until, ten weeks after the operation, she miscarried, with so great a loss of blood as to induce syncope and extreme debility, after which the condition of the eye immediately and rapidly improved; and in Mr. Dalrymple's case, severe hæmorrhage occurred twice from the wound, which, though dangerous at the moment, was followed by marked improvement in the eye.[1]

The third case, of Mrs. S., is valuable as showing, not only the condition of the parts within the orbit, but of the brain also after the operation. Whether the softened condition of the left hemisphere of the cerebrum, and more particularly of the disorganized portion of its middle lobe just above the aneurismal carotid, is to be set down as the result of the sudden cutting off the supply of blood, of the local effect of the ruptured and enlarged carotid, of the diseased uncontractile condition of most of the cerebral arteries, of the habitual opium-taking, or of the sudden want of it, or of all these causes combined, may be matter for speculation. But, inasmuch as convulsions of the left side, with paralysis to some extent of the right, occurred immediately the carotid was tied, we can hardly ignore the effect of suddenly cutting off the supply of blood; for though, in the two other cases now reported, and many others where the carotid has been tied, no such effects were noticed, still cases are mentioned where similar effects were witnessed,[2] and therefore such a contingency must not be altogether overlooked.

Though aneurism of the orbit is undoubtedly a formidable disease, only to be cured in the majority of cases by ligature of the carotid artery, looking at the satisfactory result where this has been done, we are authorised in holding out a favor-

[1] 'Med.-Chir. Trans.,' vol. ii, p. 12; vol. vi, p. 121.

[2] Among the cases of ligature of the carotid mentioned by Dr. Wood in the essay before referred to, I find three in which the operation was followed by paralysis of the opposite side of the body to that on which the ligature was placed.

able prognosis. So far as I know, the cases of Jobert and Velpeau in Paris, and Van Buren in America, were successful; and of the nine cases in which the operation has been performed in England, eight have been cured; and the cause of death in the fatal case can hardly be set down to the operation *per se*, but to the accidental complications in the case; the disease in the orbit may be considered as cured.

In conclusion I would merely call attention to the fact that, in all the three cases now related, the disease appeared on the left side, as it also did in the cases reported by Mr. Travers and Mr. Dalrymple. This may possibly be only a coincidence; if so, it is a curious one, that all the spontaneous cases, except the congenital one in the infant of Mr. H. Walton, have been so. The number is too small to draw any conclusion from, or do more than simply call attention to it, as well as that the majority of sufferers have been women; the cases resulting from direct violence have appeared in either orbit, according to the direction of the injury, and, as might have been expected, have occurred most frequently in men.

P.S.—As a fourth case of spontaneous aneurism of the orbit has recently fallen under my care, it appears desirable that it should be here related.

Case 4.—August 21st, 1859, Mrs. J—, of Kirkstall Road, Leeds, was brought to me. She is forty-two years old, and mother of seven children, the last being born only seventeen days ago. A week before that event (July 28th) she got up as usual early in the morning, but on account of great and peculiar pain on the right side of the head, "quite different from headache," which caused her to feel very ill, she had gone to bed again. While lying there, she suddenly—"sudden as a flash of light"—called out to her mother, who stood by the bed, "The pain has gone into my right eye; it feels all on fire!" The eyeball felt stiff, hot, and could not be moved. The eyelids closed, and she has not since been able to open them, though when opened by the fingers she could at first see very well. She immediately became sick and faint from the pain, noise, and confusion, over the right side of the head and in the ear. It is doubtful if there were any protrusion of the ball.

On August 4th, she was delivered of a living child, at the full term. The labour was natural, but rather hard. Since then the protrusion of the eye has rapidly increased, and the pain has been most distressing, more especially during the last few days.

At this date, August 21st, I found the *right* eyeball very greatly protruded, the sight altogether gone, the cornea becoming hazy, but not in a sufficient degree to account for the loss of sight; the pupil moderately dilated and immoveable; the lids rather livid, and so greatly distended as to be unable to meet, the intervening space being filled with a thick,

protruding, transverse fold of intensely congested chemosed conjunctiva. There was great heat in the part, and a feeling as though the eyeball would burst. Over the right side of the head and in the ear was a constant noise, "just like and as loud as the steam-hammer of the foundry." (She lives close by a large forge.) This rendered her bewildered and confused in mind, and was made worse by any hurry or excitement. There was no fluctuation, and the tumour was too solid for either suppuration or fluid. With the ear laid upon the eye, a distinct bruit could be heard, and some pulsation be perceived, but neither in nearly the marked degree they were in Cases 1 and 3: apparently the cause of both was deep and powerful, for both could be perceived over the left brow and temple. Mr. Busk, being in the neighbourhood, gave me the benefit of his opinion, and agreeing with me as to the character of the affection, as well as the propriety of tying the carotid, assisted me at the operation, which was performed on the 24th; and he also watched the case for some days afterwards. Not the slightest effect was produced upon the brain or circulation by the tightening of the ligature. The bruit and pulsation instantly ceased, the noise and distress in the head were lost, the protrusion of the ball became less, and the congestion diminished.

September 16th, twenty-three days after the operation.—The ligature came away. The lids are much cooler and smaller, the conjunctiva less vascular, the protrusion of the ball decidedly less, and the cornea is now perfectly clear, but there is not the least perception of light.

October 10th.—There is no œdema in either lid, but little heat or vascularity in the conjunctiva or globe; this is fast receding into the orbit. The cornea is quite clear, and the lens is less muddy. The texture of the iris looks natural; the pupil is dilated, as before the operation, and quite motionless. She can now just discern light from darkness. There is numbness over the brow, no power of moving the eyelids, and the ball is perfectly stationary during every effort to move it. However much the left eye is rotated, not the least movement is perceptible in the right.

There must be considerable pressure upon all the nerves of the orbit, probably in the cavernous sinus, as it is scarcely possible, if it were in the orbit, that it would equally involve every nerve, and paralyse every muscle. There certainly has been very great effusion within the orbit, to the production of nearly complete exophthalmia; but as this has so greatly subsided, it can hardly account for the total want of action in the whole of the orbital muscles, more especially as the loss of motion preceded the great effusion in the orbit. I therefore consider the pressure upon the nerves and ophthalmic vein to be in the cavernous sinus, and that it is caused by an aneurismal rupture which took place on the sudden accession of the complaint, causing by compression of the nerves and blood-vessels the distressing symptoms. Ligature of the carotid having arrested further escape, and allowed consolidation to take place, absorption of the effused blood is now going on. From the progress made it is highly probable, the whole of the effused material will be removed, and the ball will return into its normal position. Whether vision will be restored is doubtful; but considering that the cornea is now perfectly normal, the lens is less muddy, the vascularity of the sclerotic is nearly gone, that there is now a faint perception of light, and the globe is receding into the orbit, it is possible some sight may be restored, or at any rate the power of movement of the ball may be regained.

It will be noticed that this, like all the recorded cases of spontaneous orbital aneurism, occurred in a woman, but that the right and not the left side was involved.

CASE

OF

ANEURISM OF THE THORACIC AORTA,

WHICH OPENED INTO THE

TRACHEA AND LEFT BRONCHUS;

AND IN WHICH HÆMOPTYSIS OCCURRED FOUR YEARS AND EIGHT MONTHS BEFORE DEATH:

WITH

REMARKS ON THE CIRCUMSTANCES ATTENDING THE RUPTURE OF ANEURISMS, ESPECIALLY ON MUCOUS SURFACES.

BY

W. T. GAIRDNER, M.D., F.R.C.P. Edin.

COMMUNICATED BY

DR. MURCHISON.

Received April 23d.—Read April 26th, 1859.

The subject of the following observations was Mr. J. B—, a merchant, of a robust frame, and more than average intelligence, who, at the period of his death, was about forty years of age. I was first consulted by him on the 9th of March, 1854, and he remained more or less constantly under my observation till his death, which occurred on the

19th of April, 1858. The mass of details regarding his symptoms, which came to my knowledge during this period of four years, would occupy much more space than can be given to them here, and I must therefore bring within very narrow limits the statement of many facts to which the intelligence of the patient, and the anxieties of his relatives, lent much interest at the time of their occurrence.

From many conversations with the patient, I am enabled to carry back the history of his complaint to nearly six years before the date of my first visit to him, *i.e.* to ten years before his death. The earliest symptoms were pains in the left side and shoulder, sometimes pretty severe, but without any distress of breathing or palpitation. He was at this time accustomed to take a good deal of exercise, and used to experience relief from his pains on walking till he was thoroughly heated. He also found that the use of stimulants frequently removed the pain, which was always lessened after a free perspiration; and partly, no doubt, on this account, the pains were regarded as "rheumatic." He afterwards became subject to more or less of difficulty of breathing, and, under the advice of his ordinary medical attendant, underwent a considerable amount of active treatment.

In October, 1852, it was for the first time intimated to the patient that there was a fault in the great vessels, indicated by a murmur, of the precise character and site of which I have, however, no information. He was forbidden to use severe exertion, which up to this time he had permitted himself without restraint; he was also directed to use palliative remedies, and was allowed moderately good diet, with a proportion of stimulants. Previous to this time, but at what precise period in the history of the case I am unable to state, he was sensible of two distinct aggravations in his complaint, one after a fall in shooting, the other connected with an injury which he received while descending from the top of a stage-coach. After this he always acted on the advice he had received, and was very chary of exposing himself to the least risk of injury.

In March, 1853, a pulsation became apparent in the upper part of the left front, and in the course of the summer he became subject to pretty severe cough. With the cough came a mucous expectoration, and though nothing that was distinctly of blood colour was observed, the sputum was slightly coloured, or "bilious" as he called it.

It was in August, 1853, that the first gush of blood occurred. The circumstances were remarkable, and served to make known Mr. J. B—'s precarious state of health to a wide circle of his townsmen. He was giving evidence as a witness in a court of justice, under some degree of mental excitement, when, without the least warning, his mouth suddenly filled with blood; and apparently without either coughing or vomiting he brought up such a quantity of pure blood as made him quite faint. He was immediately conveyed home, and there the hæmorrhage was repeated to nearly the same extent as before. For several weeks after this occurrence Mr. J. B— was confined entirely to his room, and for the most part to the sofa; he also took sedative remedies to a considerable extent. After a time, however, being dissatisfied with the results of treatment, he changed his medical advisers more than once; and ultimately placed himself under my care, as already stated, in the spring of 1854, about six months after the first hæmorrhage, and rather more than four years before his death.

It is unnecessary to go in detail into the history of the case after this. I found the patient with every sign of a large aneurism of the aorta presenting itself in the left front immediately below the clavicle, and passing backwards and upwards so as slightly to involve the left subclavian artery. The radial pulse on the left side was a little weaker than on the right (this sign became afterwards much more distinct, the pulse being ultimately almost entirely suppressed). There was less of dulness on percussion than of tumour and impulse; there was only a trace of the murmur formerly heard. For the rest, the patient was in tolerable condition, sleeping better than he had done for some time after the serious accident of the preceding August; suffering

little positive pain, and having had no considerable expectoration of blood, although he had had a slight cough, with occasional coloured sputa. The pulse was ordinarily 70 to 80. He at this time took some exercise, but as yet very little. His hopes of life had obviously been deeply shaken, and his behaviour was that of a man doomed to a speedy and probably a sudden death.

I directed him to take gentle exercise, and after a time to go for two hours daily to his place of business, but not to lean over the desk; to take light, nourishing, solid food; to abstain from medicine; now and then, when pain was severe, to put one or two leeches over the tumour; and in general to think as little as possible about his complaint, except in the way of caution against violent movement. Under this regimen his condition improved considerably. In the beginning of May he removed to the seaside, and before the end of the same month he took lodgings at a quiet railway station on the Gala Water, on the banks of which he spent the summer with very considerable enjoyment. He became a brother of the angle, and with the help of his wife and a light rod, managed to do a considerable amount of execution among the Gala trout. His mind, too, found great relief from suffering in a resigned and intelligent view of his condition. From having abandoned all idea of an ultimate cure he seemed to derive decided comfort as regards the present no less than the future.

But in the midst of this improved state of health, he continued occasionally to bring up a more or less tinged expectoration; sometimes rusty, sometimes purple, almost never of anything approaching pure blood. This, I believe, continued to be the case, with intermissions of, at most, a few weeks, during the remainder of his life. The two succeeding summers were spent, in part at least, on the Gala; but, although the progress of the disease was slow, I had no difficulty in recognising a distinct progress, chiefly in the direction of the left lung, of which the sounds gradually became more and more impaired. Occasionally he had attacks of severe pain, and now and then paroxysms closely

resembling *angina pectoris*. He had also one or two attacks during the night, which, according to the description, I believe to have been of an epileptic character, but which lasted only a few minutes, and were not accompanied by marked convulsions. In the winter of 1857-8 it was evident to me that the end was approaching; the patient was worn and haggard in appearance; he had lost flesh to a great extent; he breathed with difficulty, and had a somewhat hoarse, rather laryngeal, inspiration, and an altered voice; he occasionally complained of difficulty in swallowing; he frequently brought up blood rather more copiously than he had done since the first attack of hæmoptysis, but never in large quantity, or pure. He also had sickness of stomach to a distressing extent, often lost his sleep at night, and was altogether in a pitiable condition. At length the respiration of the left lung became completely suppressed, the percussion at the same time becoming dull all over the left side; and on the 19th of April, 1858, a small gush of blood, probably not exceeding eight or ten ounces, occurred, which terminated life by suffocation in a few minutes.

A careful post-mortem examination was performed by Dr. Haldane; and the result of it, so far as the interest of the preceding facts extends, is now before the Society. The aneurism involves the descending aorta, from the left subclavian, which is barely free of the sac, to several inches lower down. The sac rests on the vertebræ behind, on the ribs above, on the left lung below and in front. To the left lung it is firmly adherent over a space of many inches square. The left bronchus is stretched over the sac, and has its posterior wall absorbed throughout its whole length. The sac is filled with firm coagulum, which is freely exposed to view from the interior of the bronchus. Exactly at the bifurcation of the trachea there exists another opening into the sac; it is not larger than will admit a probe, and from its smooth rounded edges has evidently been a long time present. This opening, too, rests upon a firm, solid, laminated clot. The left pneumogastric nerve, and the corresponding recurrent, are deeply involved in the wall of the

sac. The left lung is much collapsed, and infiltrated with a considerable amount of purple blood. The right lung was quite normal, and has not been preserved.

I am led to believe the preceding case not unworthy of the attention of the Medical and Chirurgical Society—1st, because of the very long period which existed between the occurrence of rupture of the sac and the ultimate fatal event; and, 2dly, because of the occurrence of hæmorrhage, in a modified form, at intervals during the whole of that very long period. There is not much room for remark on the diagnosis, which, from the time that I first saw the case, was too clear to admit of a doubt. Nor did the case illustrate any new symptom or combination of symptoms in thoracic aneurism, such as to call for remark. But as it presents an undoubtedly very rare, if not unique, example of the prolongation of life after rupture of the sac, and as this particular class of cases has not been made the subject of any very formal investigation, though incidentally discussed by most authorities, I have thought it desirable to accompany the statement of the facts with a short commentary, illustrative of some points which may have escaped the attention of members of the Society.

It is well known that sudden death by hæmorrhage in the case of aneurisms of the thorax and abdomen, opening on mucous surfaces, is very generally preceded by minor discharges of blood, and sometimes by more or less considerable gushes, occurring many hours, and sometimes many days or weeks, before the fatal event. This fact, indeed, though apparent to all who have studied the records of aneurism, was not duly appreciated until a comparatively recent period; these slight hæmorrhages being often either allowed to pass unnoticed, or being referred to some other cause than the true one. The celebrated case of Mr. Liston probably did much to diffuse among the members of his own profession a knowledge of the variable character of the hæmorrhage from aneurismal sacs, and of the remarkable intermissions to which that symptom is occasionally subject. It is well known that between July, 1847, when

hæmoptysis occurred to the extent of many ounces, and October of the same year, when cough recurred, accompanied with a rust-coloured expectoration, Mr. Liston enjoyed an almost complete immunity from symptoms; an immunity so remarkable, that all the warnings of his medical advisers were inadequate to lead him to consider himself an invalid. Mr. Liston died in December, 1847, five months after the first gush of arterial blood, and without any new considerable hæmorrhage. The trachea was found perforated in three or four places, and portions of the aneurismal clot were discovered projecting through the openings, and partially blocking them up. It is evident, therefore, that in Mr. Liston's case the rupture of the aneurismal sac had actually taken place at least five months before death; and that, notwithstanding the unclosed openings, no discharge of blood such as to cause serious alarm to the patient himself had occurred in the interval.[1]

Mr. Liston's case was probably not unique even at the time at which it was published. Very few recorded cases, however, exist in the literature of medicine, in which death was postponed so long as five months after a serious hæmorrhage from a mucous surface in aneurism. In proof of this assertion, I may mention that Dr. Edwards Crisp's laborious collection of cases, added to my own reading, and to a list with which I have been kindly furnished from the manuscripts of Dr. Sibson, whose very elaborate and careful study of this subject is known to the members of the Society, furnish in all only nine, or perhaps ten, instances in which an interval of a month or more existed between hæmoptysis and death, and in very few of these was there an interval nearly so long as in Mr. Liston's case between a considerable bleeding and the fatal event. In one case, indeed, pointed out to me by Dr. Sibson's manuscript,[2] hæmoptysis

[1] See 'Lancet' for December, 1847, p. 633.

[2] Guy's Hospital Museum, Preparation 1489[50]. I have had an opportunity to-day (April 25th) of inspecting this preparation, along with Dr. Sibson. The aneurism forms a three-fold dilatation, involving the greater part of the arch, and communicating by separate openings with

is stated to have taken place seven years before death; the connexion of the hæmorrhage with the aneurism, however, does not appear to be clearly made out by the history. Of all intervals less than a month the examples appear to be numerous enough.

It has been my fortune to observe and describe two cases of aneurism in which copious bleeding occurred on a mucous surface, and in which the interval between the first bleeding and death greatly exceeded that observed in the case of Mr. Liston. One of these cases is the one just narrated; the other was a case of aneurism of the superior mesenteric artery, remarkable as a specimen of a rare disease, and a curious, though incorrect, diagnosis, but still more remarkable as showing a minute opening (from the aneurismal sac into the duodenum), which was nearly closed at the time of the patient's death, but had led to profuse hæmorrhage twenty-two months before the fatal rupture into the peritoneum. The whole facts connected with this case are of great interest; but I cannot venture on an abstract of them in the present paper. They will be found in the 'Monthly Journal of Medical Science,' vol. x (for 1850), p. 83.

When aneurisms open into serous cavities it is rare to find death long delayed. Dr. Stokes, however, has referred to one case, where some days probably intervened between rupture into the pericardium and death. I have seen one instance in which "hæmorrhagic pericarditis" was caused by aneurism, and where I was led at the time very strongly to suspect that what appeared to be an inflammatory effusion was in reality a hæmorrhage, the blood having been churned about in the course of the movements of the heart so as to

the ascending and the descending aorta. The anterior part of the aneurism is extensively adherent to the right lung, while the posterior sacculated dilatation communicates with the left bronchus. The patient was a woman of fifty-four years of age. She had suffered for some months from palpitation, with pain between the shoulders, at times extending down the left arm; but "it was ascertained after death that she had been the subject of hæmoptysis six or seven years before." She died of hæmoptysis shortly after admission.

decolorize its fibrin. In a case of aneurism fatal by rupture into the peritoneum, also, it has occurred to me to observe adhesions which appeared to have confined the hæmorrhage, during some days at least, to the posterior part of the sac. But these cases are extremely few.

Several cases have been recorded in which, in aneurisms opening on the external surface of the body, a considerable interval existed between the opening and the fatal event. One of the most curious of these is a case communicated by Mr. Ramsay, surgeon at Broughty Ferry, to Mr. Syme.[1] A man affected with aneurism of the arch and of the innominate artery, lost a very large quantity of blood from a rupture opposite the cartilage of the third rib. The stream of blood is reported as being "somewhat larger than a quill;" and, strange to say, the patient, "nothing alarmed, got hold of a bowl, and held it at arm's length to receive the red arch, which he supposed was the contents of a 'bloody boil,' pressing the tumour with his chin to effect a more speedy clearance. After about a quart of blood had gushed out, he fainted, and the bleeding stopped." The patient lived for four months, without any new bleeding, and died in the end, not of the aneurism, but of "typhus fever." Not less curious is a case communicated by Dr. Neligan to Dr. Stokes.[2] A ship-carpenter, aged 56 years, had all the signs and symptoms of an aneurism of the aorta, opening externally about the second rib on the right side, in front. For more than a year the tumour discharged at intervals, sometimes copiously and in a continuous stream, a quantity of blood sufficient to cause alarm, and occasionally arrested with difficulty. Two of these hæmorrhages occurred under Dr. Neligan's observation. After the first of them, which was by far the most considerable, the tumour diminished considerably in size, and became much more dense, losing the fluctuating character it had previously had. The cough and dyspnœa also sub-

[1] 'Monthly Journal of Medical Science,' vol. x, p. 89.

[2] 'Diseases of the Heart and Aorta,' p. 582.

sided, and three weeks after the last hæmorrhage the patient left the hospital, "stating that he felt quite well." This is assuredly one of the most remarkable cases on record. The only flaw in it, as an observation, arises from the very circumstance that makes it so interesting, viz., that no opportunity occurred of ascertaining, with absolute certainty, that it was an aortic aneurism. I agree, however, with Dr. Stokes, in thinking that the early signs and symptoms leave no reasonable doubt of this diagnosis. And it is possible, therefore, though perhaps hardly probable, that this case may have ended in a more or less permanent cure of the disease; or, at all events, in long-continued exemption from external hæmorrhage, and death from some other cause.

Cases are not very uncommon, in which aneurisms of the aorta, after opening on one mucous surface and leading to hæmorrhage, are actually fatal by hæmorrhage in another direction, or by some other complication. I have several times seen an aneurism open nearly simultaneously into the trachea and œsophagus; the fatal event having been probably delayed for days after both openings. Similar cases have been recorded, and preparations illustrative of such double openings will be found in many museums. Rupture of an aneurism into the œsophagus, and into the alimentary canal generally, is probably rarely detected until the hæmorrhage is very large, indeed fatally large; because small bleedings, discharged downwards into the stomach and intestines, are almost sure to pass unnoticed. Hence we rarely obtain the opportunity of observing closely the process of rupture on the œsophageal mucous membrane. In the air-passages it is different; very small discharges of blood being here easy of detection. Sometimes, indeed, the true nature of the disease is overlooked, and the hæmorrhage is ascribed to pneumonia, pulmonary hæmorrhagic condensation, malignant disease of the lung, or, perhaps quite as commonly, to ordinary tubercular phthisis. I have seen each of these mistakes made by physicians nowise incompetent or inattentive; indeed, where large gushes of

blood occur in connexion with obscure physical signs, it is not unfrequently all but impossible to decide between aneurism and phthisis, unless the history, age, and appearance of the patient constitute a ground of decision. Even these grounds of diagnosis, carefully investigated, sometimes fail; and I have notes of one case, where large quantities of cod-liver oil were ordered, not unreasonably, nor without benefit, to an aneurismal patient, under the idea of his being consumptive, for several months before the true diagnosis was made.

But the cases in which aneurism is most apt to be overlooked after rupture of the sac are those in which no large hæmorrhage occurs; but in which, for weeks together, perhaps for months, an inconsiderable leakage occurs into the air-passages, assuming the form of—1st, a frothy bronchitic sputum *streaked* with blood; 2d, a rusty sputum very like that of pneumonia, but usually more abundant, more frothy, and less viscid; 3d, a deeply dyed purple or brownish-purple sputum, like the so-called "prune-juice" expectoration, characteristic of the third stage of pneumonia, and of certain forms of pulmonary hæmorrhagic condensation from valvular disease of the heart; 4th, any of the preceding, alternating with small discharges of pure, unmixed, but generally imperfectly coagulated blood. All of these forms of expectoration I have seen on several occasions; most of them occurred at different periods in the preceding case. In general, it may be remarked, that the bronchitic varieties of sputum, either stained or streaked in different proportions with blood, occur chiefly in tumours pressing directly on the trachea and larger bronchi, and not producing consolidation of any part of the lung; while the "prune-juice" sputum, and the varieties more truly resembling the expectoration of pneumonia, occur when the lung is directly involved in the tumour; or when by long-continued flattening of a bronchus, perhaps with extensive ulceration of its mucous membrane, and necrosis of its cartilages, secondary changes have been induced in the pulmonary texture. Such changes are rarely of the nature of inflammation, at least in the first instance. They partake more of the nature of collapse of the air-cells, which is some-

times the result of mere pressure on a bronchus, producing obstruction to the evacuation of the bronchial and pulmonary excretions; while occasionally we find a chronic and continuous infiltration of the lung with blood, either from the aneurismal sac through the bronchus, or from rupture of the aneurism directly into the pulmonary air-cells. The "lobular" character of these lesions is often very clearly demonstrable, and shows that they spring, not from inflammation proper, but from some derangement of the mechanism of the air-passages. In the more advanced stages, however, inflammatory changes are apt to occur; and I have repeatedly seen an entire lung, or some considerable portion of it, ulcerate and break up into suppurating cavities, under the continued pressure of an aneurism upon the bronchus. In one or two cases this has been attended with many of the symptoms and signs of tubercular ulceration, and the morbid appearances after death have also to a remarkable degree resembled those of softened tubercle; confined, however, absolutely to the side on which the aneurismal pressure took place. Possibly some of the cases alleged to be "aneurism associated with phthisis," may have been of this kind.

In systematic works, it is customary to ascribe the minor hæmorrhages to which I am now alluding, not to rupture of the sac, but to "congestion of the lung" from pressure on the veins, and consequent impediment to the return of blood. I am certainly not prepared to maintain that the pressure of aneurisms on the pulmonary veins never causes hæmorrhage. But that this is the chief cause of minor hæmorrhages cannot, I think, be admitted; 1st, because even in cases in which pressure on the veins may have occurred, hæmorrhage (at least continuous or repeated hæmorrhage) is almost always associated either with direct pressure of the sac upon the lung, or upon an ulcerated bronchus; 2dly, because some of the most characteristic cases I have observed of slight and continuous hæmorrhage have been from aneurisms in which no pressure on the pulmonary veins was possible; but in which there was undoubtedly pressure upon, and opening of the sac into, the trachea. I may refer in

particular to two cases of aneurism with laryngeal symptoms, which I recorded some years ago with a view chiefly to other points of interest, but in one of which the patient died of dyspnœa after weeks of constant though inconsiderable hæmorrhage; while in the other a fatal hæmorrhage occurred after many days of very slight tinging of the sputum. In both these cases there was no reasonable doubt, from the condition of the mucous membrane of the trachea, that the blood must have come from the sac.[1]

While, therefore, I will not venture to say that blood in the discharges of a patient affected with aneurism *always* indicates the communication of the sac with a mucous membrane, I believe it *generally* does so; and more especially is this the case in aneurisms accompanied by hæmoptysis, if the pressure of the tumour be on the trachea, and if it be unaccompanied by the indications of pulmonary change. The importance of this view, if correct, both as regards the diagnosis of obscure cases of aneurism, and the prognosis of this disease in well-marked cases, it is unnecessary to point out at length. I may be permitted, however, to detain the Society over one view of diagnosis, which, if it be as generally applicable as my own experience would lead me to affirm, must be one of considerable importance. In aneurisms characterised chiefly or exclusively by laryngeal symptoms, it is often extremely difficult to arrive at a satisfactory conclusion as to the cause of the very distressing dyspnœa. Now, it is precisely in this class of cases that the repeated presence of even small quantities of blood in the sputum becomes a most valuable means of diagnosis. For, if there be laryngeal dyspnœa and stridulous respiration (which are seldom present to any marked extent in mere laryngeal phthisis); if the epiglottis be not thickened; if the mucous membrane of the larynx, in so far as it is within reach of the finger, be sound; and if, with these signs, positive and nega-

[1] See 'Monthly Journal of Medical Science,' vol. xiii, p. 137; and vol. xvi, p. 116.

tive, there be a persistent tendency to even the slightest amount of blood in the sputum, while auscultation and percussion give negative results both as regards the lungs and heart, I believe that aneurism may be predicated with as near an approach to certainty as is possible without the physical signs of tumour; and further, the aneurism will be small; it will arise from the back part of the arch, or from the commencement of the innominate artery; and it will be so placed as to entangle either the left or the right recurrent nerve. These considerations have more than once led me to the diagnosis of aneurism under circumstances where, without them, it would have been impossible to give a decided opinion; and hitherto they have not led me wrong. In fact, there is but one form of disease which, in any considerable number of instances, leads to laryngeal stridor and to hæmoptysis, without positive ulceration of the larynx. Malignant tumours very closely resemble aneurism in their diagnosis in these respects. But I have never yet seen malignant disease of the chest leading to hæmoptysis, without manifest physical signs of disease in one or other lung. So that I am inclined to believe that the rules of diagnosis mentioned above, will hold good in the great majority of instances.

A reference to a drawing[1] illustrating the usual mode of opening of an aneurism upon a mucous membrane may close this rather desultory paper. It is from the case of a man who died, not of hæmorrhage, but of suffocation; but in whom, nevertheless, an opening into the trachea existed, which had yielded blood in small quantities for some time before the fatal event. The rupture has been arrested by death at its earliest stage; and it will be observed that while five or six minute papillary eminences, with distinct pale apices on a congested membrane, are to be seen, only one of these has been perforated; the actual opening not exceeding

[1] This drawing was exhibited at the meeting of the Society at which this paper was read.

the size of a pinhole. The mucous membrane as viewed from within, opposite a necrosed cartilaginous ring, is puffy and thin; and in all probability a larger rent would ere long have formed there. But it is very easy to understand on looking at this drawing, how these small pinhole openings should sometimes heal up; especially after a sudden removal of pressure, such as occurs after a copious hæmorrhage. Such would appear to have been the course of events in the case of Mr. J. B—, and also in that of Mr. Liston; as well as in the case of aneurism of the superior mesenteric artery referred to above.

A CASE

OF

POPLITEAL ANEURISM,

SUCCESSFULLY TREATED BY

FLEXION OF THE KNEE-JOINT.

BY

ERNEST HART,

SURGEON TO THE WEST LONDON HOSPITAL AND NORTH LONDON OPHTHALMIC INFIRMARY; LATE DEMONSTRATOR OF ANATOMY AT THE ST. GEORGE'S HOSPITAL SCHOOL.

COMMUNICATED BY

ALEXANDER SHAW, TREASURER.

Received Jan. 27th.—Read April 26th, 1859.

I HAVE ventured to bring this case under the notice of the Fellows of the Royal Medical and Chirurgical Society, under the impression that they may be interested in the successful treatment of so formidable a disease as popliteal aneurism by the simple flexion of the knee-joint.

J. S—, æt. 41, consulted me in the month of September, 1858, having a popliteal aneurism in the right ham. It was globular, of the size of a small apple, and situated at the lower and outer part of the popliteal space. It had a full beat, but was not very near the surface.

Placing the patient on the sofa, and baring the leg in

order to make a careful examination of the tumour, I found that its pulsation was affected by the angle at which the leg was bent upon the thigh, and that when very complete flexion was effected, its thrill almost wholly ceased. Concluding that in this position the course of the blood through the tumour was greatly retarded, I conceived the hope of effecting the cure of the aneurism by the deposition of active[1] clots, if the leg could be retained for a sufficient length of time in the bent position.

My patient had been taking active exercise, and I thought it desirable to enjoin a week's rest in bed before the commencement of any plan of treatment. During this time he complained greatly of pain in the right ham, which prevented him from sleeping at night. In other respects his health was good.

At the commencement of the second week I bandaged the leg from the foot to the knee, not covering the tumour, and having thoroughly flexed the leg on the thigh, retained it in that position by the application of a stout roller. He was a thin, wiry man, and the flexion produced no inconvenience to him at the time. On the same evening, he complained that the position was not very comfortable; the bandage had become slightly loosened, and I tightened it.

Next morning I found that he had slept moderately well. He had passed a better night than any during the preceding week had been. The pain due to the aneurismal tumour was considerably lessened. He felt some pain referred to the knee-cap, and occasioned by the flexion; but he said that this was "very trifling, and barely deserving to be called pain." I pressed my index-finger into the angle of the ham, and could not detect any pulsation. On the evening of the second day his condition was unaltered.

On the morning of the third day he had passed a restless night, but free from the pain which he used to experience. He complained of pain at the knee-cap, and of the skin feeling tight, as though the knee were too much bent. I unloosed the bandage and slowly moved the leg to a right

[1] See Broca, 'Des Anévrysmes.'

angle with the thigh. This relieved him of that sensation. I could now examine the tumour, which felt hard and partially solidified. I could detect a faint thrill, but there was none of the strong throbbing which was present in the first instance. At night I again fastened the leg in its former position.

On the fourth day his condition was most satisfactory, and did not call for any change. I could not detect any pulsation by inserting my forefinger into the angle of the ham.

On the fifth day he was somewhat restless, had not slept much during the night, and there was some pain at the knee-cap. He was cheerful and of good appetite.

I removed the bandage, and again drew down the leg to about a right angle with the thigh. The tumour was now hard and solid, and I could not detect any pulsation.

I allowed the limb to remain in this position during the day, with a light bandage fastening the foot to the thigh.

Next day I found the leg at a somewhat obtuse angle, the patient having slightly lowered it, and in consequence of the relaxation of the bandage. There was not any pulsation in the tumour, which continued to be hard and solid to the touch.

On the seventh day I attached the leg to the thigh at a slight angle, and allowed the patient to move about, the foot being slung.

On the twelfth day the leg was completely straightened, and the patient walked on it with ease, limping as he walked from the stiffness consequent upon the previous confinement of the knee-joint.

Six weeks subsequently the tumour was greatly diminished in size, but still hard and firm. It was then seen by Mr. Coulson. Three months after treatment the tumour was not perceptible, and there was no pulsation in this part of the artery. He was then seen by Mr. Buxton Shillitoe, who happened to be present when my patient presented himself.

During the course of the treatment he was seen by my friends, Mr. White Cooper, of St. Mary's Hospital, Mr. Holmes, of St. George's Hospital, and Mr. Flower, of Middlesex Hospital, by whom the progress of the case was observed.

The treatment by flexion in this case was perfectly and immediately successful. It was unattended with any difficulties, it offered no inconveniences, and was not followed by any other than satisfactory results.

The case, however, was one particularly well suited to such a plan of treatment. The patient was not stout, which renders flexion difficult; nor was he aged, which makes it painful. The tumour was of average size and of average prominence; when the knee was bent it was below the line of flexure. I believe that these are all favorable circumstances.

Cure is evidently effected in this method by the retardation of the current of blood, and the consequent deposition of active clots in the sac—the only manner in which satisfactory cures could be anticipated.

This result is probably effected by the combined influence of pressure on the sac and acute flexion of the artery.

In so far as it is due to pressure, it appears to be a return to the old method of treatment by direct pressure, but it is free from the inconveniences of the screw and pad, which were open to the reproach of occasioning gangrene of the skin, rupture of the sac, and other accidents.

Its simplicity, and its success in this case, appear to strongly recommend it for further trial. If it be not always successful, there is not any other method free from the same objection; and there appears reason to hope that this principle may admit of efficient application to other cases in which aneurismal tumours are developed opposite to the joints of the limbs.

A CASE

OF

POPLITEAL ANEURISM,

SUCCESSFULLY TREATED BY

CONTINUED FLEXION OF THE KNEE-JOINT.

BY

ALEXANDER SHAW, TREASURER,

SURGEON TO THE MIDDLESEX HOSPITAL, AND LECTURER ON SURGERY.

Received Jan. 27th.—Read April 26th, 1859.

W. T—, æt. 30, a house-painter, was admitted into the Middlesex Hospital, November 30th, 1858, for a pulsating tumour in the popliteal space of the left leg. Six years ago he had rheumatism, which lasted for four months: otherwise, his health has always been good. A week before his admission he began, without any apparent cause, to feel pain in his left knee. Four days afterwards, from slipping on a piece of cabbage leaf, he strained his limb, and the knee became swollen and more painful.

On the following day, which was Sunday, he thought to remove the stiffness by taking a walk; but the pain and lameness were so much aggravated by the exercise that he had difficulty in returning home. He rested his leg on Monday, and on Tuesday he was admitted into the hospital.

The tumour was as large as a common-sized lemon; it

completely filled the popliteal cavity, and was slightly prominent behind. It pulsated with much force: when grasped on each side, the fingers were expanded by the dilatation of the tumour as it filled; by compressing the femoral artery the pulsation was arrested: the peculiar *bruit* of aneurism was audible: the pulsation in the tibial arteries at the ankle-joint was perceptible, but obscure; there was an absence of œdema in the leg, and the temperature was natural.

It was considered, from the rapid growth of the tumour, the compressibility of its walls, and the distinctness of the pulsation, that the parietes of the sac were thin, and the contents chiefly fluid blood.

December 1st.—Having received information that a case of popliteal aneurism had lately been treated successfully, and the cure effected within a short period, with little suffering to the patient, by Mr. Ernest Hart, through adopting the simple measure of fixing the knee of the affected limb in the bent position, I resolved to give a trial to that treatment in the present case. The patient was directed to lie on his left side, and, with his thigh drawn up towards his body, to bend the knee-joint to the utmost; the calf of the leg was thus brought closely in contact with the back of the thigh: a broad strap was now coiled about the foot, then carried round the thigh near the hip, and fastened with a buckle. When the limb was secured in that manner, the finger, previously oiled, was inserted deeply into the angle behind the knee; but no pulsation could be perceived; nor was the patient sensible of any beating in the tumour, such as he had been lately accustomed to. In both tibial arteries near the ankle-joint the pulsation was so faint that it was doubtful whether it could be felt.

2d.—His sleep was broken last night from the pain and irksomeness of having his knee bound up; the chief pain being in the line of the ligamentum patellæ.

On the outside of the knee there was slight redness from pressure; an amadou plaster was applied to protect the skin.

3d.—Although he still complains of pain in front of the knee, and of his rest being disturbed, yet he looks fresh and well; and the night-nurse reports that he sleeps soundly for several hours consecutively. The strap was a little slackened.

4th.—Each day the finger has been introduced into the popliteal space, as far as the bent position of the knee would permit, to feel for pulsation; but none has been perceived. To-day the strap was removed, and the knee partially unbent, so that the tumour could be accurately examined. At once a decided improvement was manifest; the tumour had lost about one third of its original size; its walls were solid and incompressible, and the pulsation, compared with what it had formerly been, was feeble and indistinct. As the tumour had now receded into a deeper part of the popliteal cavity, before fixing the knee again in the bent position, a glass marble, an inch in diameter, covered with wool, was inserted into the hollow, to assist in compressing the artery and sac.

8th.—Since last report several examinations have been made by undoing the strap; but owing to the stiffness of the knee from having been kept constantly bent, the popliteal cavity could not be opened out sufficiently for ascertaining the true condition of the sac. However, yesterday the tumour appeared so much reduced in size, its walls so dense, and the pulsation so faint, that it was thought that a trial might be made of discontinuing the flexion. The strap was accordingly removed, and the patient instructed to straighten the limb as much as he could. But the result has been disappointing. Although the knee is still stiff and a good deal bent, yet the tumour can be felt to be more prominent, and the pulsation stronger, than was supposed to have been the case. It was, therefore, resolved to keep the knee fixed in the bent position for a longer time. The patient readily consented; for, although he has not lost the pain in front of his knee, and there is even slight heat and swelling at that part, for which a lead lotion is applied, yet he has shown no reluctance to continue the treatment; indeed, he

himself willingly superintends the use of the strap, and draws it with sufficient tightness round the limb to secure the knee in its greatest state of flexion.

January 7th, 1859.—It has not been thought necessary to insert the reports made at different dates since the last; for the purport of all has been nearly the same. Ten days subsequently the treatment was varied by removing the strap for some hours each day; or by taking it off during the night, and replacing it in the day. From relaxing the limb in that manner the patient obtained a little relief; but owing to the stiffness of the joint the knee remained, when liberated, nearly as much bent as before. In the examinations made at that time, the sac was observed to become gradually smaller and denser, and the pulsation to grow fainter. Between the third and fourth week from the commencement of the treatment, the pulsation was so feeble that it was daily expected to cease. It continued, nevertheless, to be felt; and eight days ago, with the hope of effectually stopping it, the patient was instructed to keep the knee for the four following days tightly bound. When the joint was released, at the expiration of that time, the beating in the tumour, though extremely faint, was not extinct; and it could be discerned yesterday. But this day, being the thirty-eighth from the commencement of the treatment, on the most careful examination of the tumour, not the slightest pulsation can be detected.

11th.—The pulsation is no longer felt. The tumour is hard, and of the size of a walnut. On examining the anterior and posterior tibial arteries, a very faint, almost imperceptible pulsation could be detected in the former, but there was none in the latter. Around the knee several anastomosing vessels could be felt beating. The patient is slowly regaining the power of straightening the knee. To-day he was allowed to get out of bed.

14th.—There has been some œdema of the left leg, from sitting with his feet down. He walks with difficulty, from not being able to extend his knee.

19th.—The stiffness in the knee is gone, and he has only

a slight halt in his gait. The tumour, so far as it can be distinguished from the surrounding condensed structures in the ham, is of small dimensions. The pulsation of the anastomosing vessels is distinctly felt in various parts over the joint, and is attended with a faint thrill, perhaps from their being tortuous. The temperature of the left knee is perceptibly higher than that of the right.

25th (the fifty-sixth day).—He was discharged cured.

Remarks.—In addition to what has been already said of the pain attending the confinement of the knee in the bent position, it may be stated that, on the whole, it was not considerable. After the first fortnight the patient ceased to complain, and he did not ask, at any time, to have the belt relaxed.

No particular system of diet was enforced.

In regard to the principle on which the cure by the above mode of treatment was effected;—it appears that three distinct causes co-operated to diminish the force of the circulation through the aneurismal sac, and thereby favour the deposition on its inside of successive layers of solid fibrine. First, by the bent position of the knee, the whole tumour was subjected to a general and equal compression within the popliteal cavity. Secondly, the mass of the tumour, confined in the limited space of the ham, would act as a compress or pad upon the artery, to diminish its calibre. Thirdly, chief importance is to be attached to the bent or doubled condition of the arterial trunk, consequent on the acute angle at which the knee-joint was kept flexed. It is, I believe, generally known that if a person bend his elbow-joint to the utmost, the pulse at the wrist will be stopped. By numerous trials on different individuals, I have satisfied myself that when the knee is similarly flexed, the pulsation in both tibial arteries at the ankle is arrested; and I have remarked that after the limb had been straightened, it took from three to five seconds for the pulsation to be restored.

ON

SOME OF THE EFFECTS

OF

PRIMARY CANCEROUS TUMOURS

WITHIN

THE CHEST.

BY

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Cancerous tumours in the chest have long been divided into two classes—secondary cancerous tumours and primary cancer.

Secondary cancerous tumours, the result of cancer originating in a remote part of the body, are common, and are usually multiple and scattered through both lungs.

Primary cancer within the chest is comparatively rare, and usually forms a single compact nodular mass, commonly occupying the mediastinum, and invading to a greater or less extent one of the lungs—in the great majority of instances, the *right*—the other lung remaining uninfected.

Secondary cancerous tumours scattered through the lungs have no special tendency to set up inflammation in any of

the neighbouring tissues, and are often found of considerable size with the pulmonary tissue immediately surrounding them perfectly healthy.

Primary cancer, when it involves the mediastinum and root of one lung, leads to adhesion of that lung to the pleura costalis, and, in the end, to suppurative and destructive inflammation of the tissue of the lung where it is not invaded by the cancer.

It is to the particulars of some cases of this kind that I desire to call the attention of the Society to-night.

Case 1.—Henry O—, a railway clerk, æt. 31, was admitted into King's College Hospital on the 9th of October, 1847.

He stated that his health was good till May, 1846, when he was laid up with what was considered pneumonia of the lower lobe of the right lung.

From that time he had shortness of breath on exertion, and was in the habit of spitting up in the morning a little clear phlegm.

In February, 1847, he was again laid up for a time with pain in the lower part of the right chest.

Six weeks before his admission to the hospital, he was taken again with sharp pain in the right side and between the shoulders, and became weak and languid. On admission to the hospital, though according to his own statement he had fallen away much in flesh and strength, he was still in tolerable general nutrition.

He complained of pain between the shoulders, almost constant while he was sitting up, and of occasional pain in the lower part of the chest. He had slight cough, and the matter expectorated was tinged with blood.

The right side of the chest was everywhere duller on percussion than the left, but more especially so in the lower part.

The respiratory murmur was quite inaudible over the lower two thirds of the right lung, and was very faint in the upper third.

The liver was felt extending some distance below the margins of the ribs. The pulse was 128, the inspirations 26, in the minute. He had very little appetite, a furred tongue, and costive bowels.

The pain between the shoulders was somewhat relieved by a blister, and his condition in other respects slightly improved after his admission to the hospital, but the symptoms remained essentially the same.

The pulse ranged from 120 to 132; the inspirations from 24 to 32. He spat up daily a small quantity of phlegm, which was generally tinged with blood.

On the 10th of November there was much œdema of the right side of the chest and face, and soon afterwards the superficial veins of the chest became enlarged and conspicuous. The vesicular respiratory murmur was faint, but distinctly audible, in the upper part of the right chest. The cough was very slight, and the matter spat up continued to be tinged with blood.

On December 15th it was noted that the left side of the chest was dilated, as was supposed, from the extra duty thrown on it by the deficiency of breathing in the right lung. The superficial veins of the abdomen were becoming enlarged and tortuous, like those on the right side of the chest. On the left side of the chest behind, a single enlarged tortuous vein was seen.

Up to this time the cough had been slight and the expectoration scanty.

On the 20th of December the cough had become much more troublesome, and a coarse crepitus was heard under the right clavicle.

On the 22d a loud, superficial rubbing sound was heard over the cardiac region. The expectoration was still scanty.

The friction sound in the precordial region continued to be audible up to the 31st, and was not noticed subsequently.

All this time the œdema on the right side of the chest continued. The superficial veins on the right side of the

chest behind became more and more enlarged and tortuous. The patient constantly grew weaker and more anæmic; and on the 6th of February left the hospital at his own desire.

On the 17th of February he was readmitted, in a state of orthopnœa, much more pallid and wasted than when he left, and with his body and extremities covered with purpuric spots. He expectorated a yellow puriform matter.

The right side of the belly, and the right and front of the chest, were covered with enlarged tortuous veins.

Over the precordial region a rubbing sound was heard. He died the next day from apnœa.

On examination of the body, the lower part of the right chest was found occupied by a white cancerous mass firmly adherent to the ribs and diaphragm. This cancerous mass extended in form of a cone up the mediastinum, its apex being on a level with the clavicle. The left boundary of the tumour was very irregular, posteriorly extending over the bodies of the vertebræ to the left side, displacing the aorta, which was firmly attached to it.

It penetrated the superior vena cava in nodular or globular heads, half filling its cavity and projecting into the right auricle. The root of the right lung was quite inclosed in the mass, and the large bronchi were penetrated by the cancer, and were narrowed but not closed by it.

The right lung was everywhere firmly adherent by thickened pleura to the walls of the chest. The place of the upper lobe was almost occupied by a large cavity half filled with pus. The rest of the lung not invaded by the tumour was a gray hepatized mass, containing small pockets of pus.

The outer surface of the pericardium was covered with recently effused lymph, and the layers of the pericardium were glued together by an effusion of lymph. The heart itself appeared to be healthy.

The left lung and its root were in no way implicated in the disease, and the texture of the lung was quite healthy.

In this case a cancerous tumour, occupying the lower lobe of the right lung, and extending up the mediastinum so as to embrace the root of the right lung, led to universal adhesion of that lung, by thickened pleura, to the walls of the chest; to almost entire destruction of the tissue of the lung not invaded by the cancer; and, lastly, to an effusion of lymph on both surfaces of the pericardium.

Case 2.—F. S—, a compositor, æt. 20, was admitted into King's College Hospital on the 14th of July, 1858.

He stated that he was quite well till Whit-Monday, the 24th of May preceding, when he took cold, as he imagined, from having rowed hard in the rain on the River Lea. A few days after this he felt pain on the right side of the chest behind, near the right shoulder-blade. This was soon succeeded by pain in front of the chest, near the right nipple. These pains ceased at the end of a week, and he had no further particular ailment till the 3d of July, when œdematous swelling of the face came on, with much vascular fulness, so that on any exertion his face became of a purple colour, and, to use his own expression, he was obliged to rest while the flow of blood to the face went down.

The œdematous swelling of the face continued. It was greatest on his rising in the morning, and subsided after he had been sitting up some hours. In spite of these ailments his appetite continued good, and he followed his occupation.

On his admission to the hospital, the 14th of July, there was œdematous swelling of the face, the hands were purplish, and the veins on the surface of the chest were enlarged. It was evident there was some obstruction to the course of the blood through the descending cava; and an examination of the chest led to the conclusion that this obstruction was caused by a tumour in the upper and anterior part of the chest. No unusual pulsation existed in that region, and no morbid *bruit* attending the movements of the heart could anywhere be heard.

He complained of occasional headache and nausea, but

his appetite was good; his pulse was 72—76. He made no complaint of cough, and slept well.

He suffered chiefly from the swelling of the face and neck, and from the venous distension of the face on any exertion.

No marked change occurred in his symptoms, and on the 7th of August he left the hospital.

On the 25th of September he was readmitted. The chest was then covered with enlarged tortuous veins, communicating with three or four superficial epigastric veins, which ran comparatively straight to the pubes. He had difficulty of breathing, and a husky voice; was troubled with cough; and spat up frothy mucus, often tinged with blood, and mixed occasionally with tough, puriform masses. A coarse crepitus was heard over the whole of the right lung. The respiratory murmur on the left side was somewhat harsh, but no crepitus was heard except quite at the base behind.

On the 1st of October he was taken with vomiting after eating a few grapes, and from this time onwards was much harassed with vomiting.

His condition now got gradually worse. The cough became more frequent and irritable; he expectorated abundantly viscid, frothy mucus, mixed with greenish puriform masses, and had a good deal of fever, the pulse being usually from 100 to 108. It was noted that there was no irregularity of the pupils. Creasote, chloroform, and other medicines were given to stop the vomiting, but with only partial success.

On the 22d he could not lie back on account of dyspnœa. During the following night had several spasmodic attacks of difficulty of breathing; and in the morning of the 23d he died—from dyspnœa.

On examination of the body, a yellowish-white (cancerous) tumour was found to occupy the anterior mediastinum, reaching almost to the sternum anteriorly, but separated from it by a little loose tissue, in which a few enlarged glands were lodged. The tumour was a firm,

gristly, nodular mass, about four inches long, and rather more than three inches in width. About two thirds of the mass was situated to the right of the middle line, and it invaded for about an inch in depth the tissue of the right lung.

The right lung was everywhere firmly adherent, so that it was with difficulty removed from the chest. It was granular, of a reddish-gray colour, tore readily, and the torn or cut surface had scattered over it small spots of yellow matter, which, under the microscope, proved to be pus, with some débris of the pulmonary tissue. In several places the destruction of the pulmonary tissue was so complete that small vomicæ had formed, two or three of which were of the size of a small walnut.

The left lung, which was quite free from adhesion, was much congested and strewed with points of pulmonary apoplexy—the size of pins' heads—but was everywhere crepitant.

The pericardium contained about half a pint of sero-sanguineous fluid. The heart was small and contracted. The valves were healthy.

On dissection, it was found that the superior vena cava, the whole of the right innominate vein, and a small portion of the left innominate vein, were involved in the tumour. The superior cava was so narrowed, that only a No. 1 catheter could be passed through it. At the termination of the superior cava, the cancer projected about half an inch into the pericardium, the parietal layer of which was there reflected over the tumour. The terminal part of the vena azygos was also involved in the tumour, and quite obliterated by it. The descending vena cava was much dilated, but otherwise healthy.

The right branch of the pulmonary artery also passed through the mass, and was very much narrowed by it. The trunk of the pulmonary artery and the left branch were free.

All the pulmonary veins passed below the tumour, and were not involved.

The cancerous mass rested on, and was intimately connected with, the ascending and transverse portions of the arch of the aorta, evidently narrowing them, but not penetrating their coats. The innominate artery passed through the mass, but was not obstructed by it. The left carotid and subclavian arteries passed to the left of the tumour, and were not involved.

The right pneumogastric nerve passed through the substance of the mass, entering it about the level of the upper margin of the sternum, just before giving off the right recurrent. The right phrenic nerve was not traced.

The left pneumogastric and phrenic nerves passed to the left of the tumour, and were not involved.

On opening the trachea and bronchi from behind, it was found that the cancer had penetrated the anterior part of the tube, from half an inch above the bifurcation to half an inch down the right bronchus, projecting as a nodular mass on the inner surface of the tube. A smaller cancerous nodule projected into the left bronchus, just below the bifurcation.

The calibre of the right bronchus was considerably narrowed by pressure from without, and by the cancer projecting within it. The glands situated at the bifurcation of the bronchi were enlarged.

There was no cancerous mass apart from the tumour in the lung itself.

In this case the cancerous tumour involved the root of the lung, but had invaded the pulmonary tissue only to a slight extent. The lung was found firmly adherent to the pleura costalis, and the tissue everywhere granular and friable, having scattered through it small collections of pus, mixed with débris of the pulmonary tissue. If the poor man had lived a short time longer the destruction of the lung would evidently have been as complete as in the former case. The tumour involved the pneumogastric nerve where it forms behind the right bronchus, the posterior pulmonary plexus. The right branch of the pulmonary

artery also passed through the tumour, and was much narrowed by it. The pulmonary veins were free.

Case 3.—W. W—, a coachman, æt. 63, was admitted into King's College Hospital on the 15th of March, 1848.

According to his own account he had general good health till October, 1847, when he became gradually affected with a dry, husky cough, and some shortness of breath. These ailments did not lay him up, but they continued, and the shortness of breath grew gradually worse.

He had no other particular ailments till three weeks before his admission to the hospital, when swelling of the face came on, with much vascular fulness on his stooping or leaning forward. From the commencement of his illness he had suffered no pain in the chest, experienced no difficulty of swallowing, and never lost his voice.

On his admission to the hospital there was slight œdema of the face and great distension of the jugulars—the face becoming almost purple from congestion on his leaning forward. He had a frequent, dry, husky cough.

On examination of the chest there was no projecting tumour discoverable, and no undue pulsation anywhere. There was dulness on percussion in front of the chest on the right side, and very slight vocal vibration, as compared with the opposite side, especially between the second and third ribs, and from the sternum to a vertical line through the nipple. Over the same space the respiratory murmur was scarcely audible, but both sounds of the heart were loud—louder than in the corresponding space on the opposite side.

Over the right scapula there was almost complete absence of vocal vibration.

Respiratory murmur was heard distinctly just below the right clavicle and in the lower part of the right chest. Breath sounds loud on the left side of the chest.

No morbid "bruit" was heard over the heart or the large arteries. No difference in the pulse of the two sides. No

difficulty of swallowing. Voice tolerably clear. No enlarged glands in the axilla or above the clavicles.

He was in a state of tolerable nutrition for his time of life; had a very good appetite. Pulse 74; respirations 22.

After admission to the hospital the venous distension of the neck and face increased, and on the 3d of April he began to spit blood.

On the 4th of April he spat up half a spitting-basin full of blood. He was in consequence bled from the arm to twelve ounces. The blood drawn was not buffed or cupped.

The spitting of blood ceased for a time.

On the 5th it was noted that the cutaneous veins in the lower part of the chest in front on both sides were distended and varicose.

On the 7th he expectorated bloody mucus, and had become hoarse. Pulse 104 (on sitting up, after coughing), regular; respirations 27.

The chest and arms, as well as the neck and face, were slightly œdematous.

The minute veins on the surface of the chest on both sides had become varicose and conspicuous; but he was still quite free from pain.

Soon after the last notes were taken his face became enormously congested; he complained of cold and shivering, and went to bed in a very exhausted state. During the night he coughed incessantly, and could only rest with his head supported in the sitting posture. He had no fresh spitting of blood, and died from asphyxia at 9 a.m. the following morning.

On examination after death the right pleura contained a pint and a half of serous fluid. The whole of the upper lobe of the right lung was firmly united to the pleura costalis; the middle and lower lobes were free from adhesions. When the upper lobe was dissected out it presented the appearance of a white solid tumour, but, on cutting through it, this appearance was found to be owing to an enormously thickened pleura.

The included lobe was condensed and solid, but had the colour natural to pulmonary tissue.

Behind the right bronchus, immediately below the bifurcation, was an irregular scirrhous mass, about the size of a small apple, apparently originating in the bronchial glands, almost surrounding without constricting the right bronchus, and surrounding the descending vena cava, and encroaching on it so much that, for about half an inch, it was not larger than a small goose-quill. There were a few extremely minute cancerous tubercles in the upper lobe, especially in the immediate vicinity of the tumour.

The middle lobe of the right lung was flaccid and atrophied. The lower lobe sound.

The left lung free from adhesions and sound.

The heart sound. The aorta large, presenting on its inner surface much atheromatous deposit.

The aorta and the large branches springing from it were not involved in the tumour.

There was no cancer in any other part of the body.

In this last instance the tumour, which was smaller than in the preceding cases, formed an irregular scirrhous mass, almost surrounding but not constricting the right bronchus. The changes in the lung were also less extensive than in the preceding cases. The upper lobe of the right lung was firmly adherent to the wall of the chest. The pleura covering it enormously thickened, and the lobe itself condensed and solid. The middle and lower lobes of the lung were free from adhesions. The middle lobe flaccid and atrophied, and the lower lobe sound. The inflammatory disease caused by the tumour was confined to the upper lobe of the lung.

A case very similar to those I have just related is recorded by Dr. Sims in the eighteenth volume of the 'Medico-Chirurgical Transactions.'

The patient, a baker, æt. 43, died under the care of Dr. Sims, in the Marylebone Infirmary. The cancerous mass,

in this instance, was extensively attached to the right lung and pressed upon the posterior part of the right auricle. The right bronchus, with several of its subdivisions, passed directly through it. Dr. Sims thus describes the condition of the lungs.

Right lung.—Pleura much thickened. This lung occupied a considerable space, for the augmented contents of the thorax had encroached upon the cavity of the abdomen. A great proportion of it was consolidated, apparently in consequence of old hepatization; in some parts the substance crumbled on the application of gentle pressure—this portion was of a dark or dusky-red colour; there was pus in a few small cavities in the section. A small portion was comparatively healthy, and in degree fit for the purposes of respiration.

Left lung.—"The left lung was free from adhesion, and the pleura of natural appearance." "This lung was considerably indurated in some parts, which, on being cut into, consisted of extensive red hepatization. On a careful examination of the lung it was found to be quite free from the morbid growth contained in the other cavity of the thorax; the larger vessels and the bronchi were not engaged in the disease."

The tumour embraced the descending cava, and penetrated its coats.

The right branch of the pulmonary artery passed through the tumour and was much dilated, but retained its texture.

One of the right pulmonary veins passed into the centre of the tumour and was there lost.

In all the preceding cases a primary cancerous tumour in the chest involved the root of the right lung, and remarkable changes, of inflammatory origin, were found in the chest. These changes consisted, in the order of their frequency, of—

1. Firm adhesion of the lung to the walls of the chest by thickened pleura.

2. Inflammatory condensation of the lung, where it was not invaded by the cancer, proceeding in three of the cases to more or less disorganization of the pulmonary tissue and the formation of pockets of pus.

3. In one of the cases—the case in which the tumour had attained the greatest size and spread furthest towards the left side—adhesion of the pericardium and an abundant effusion of lymph on its outer surface.

The extent of change in the lung in the different cases was greater as the tumour was larger, and involved more completely the root of the lung; and in all the cases the left lung was free from adhesions and presented no other changes than those which result from recent congestion.

It is obvious from these circumstances that the diseased condition of the right lung was the result of the cancer, and the question arises, by what agency did the cancer produce it?

Cancer has no direct tendency to cause inflammation of the surrounding tissues, and secondary cancerous tumours, scattered through the lung, are generally surrounded by perfectly healthy pulmonary tissue.

In the cases which form the subject of this paper, the cancer evidently impaired the nutrition of the tissues of the lung, because it involved the root of the lung, and thus interfered with the office of the blood-vessels and nerves on which the function of the lung and its healthy nutrition depend.

The changes of inflammatory origin can hardly be attributed to obstruction of the blood-vessels of the lung caused by the tumour. Obstruction of the pulmonary and bronchial arteries might cause gangrene or atrophy of the lung, which have been observed as part of the morbid changes in such cases, and obstruction of the pulmonary veins might cause hæmorrhage; but neither of these conditions is a probable cause of the thickening and adhesion of the pleura, and of the inflammatory destruction of the lung which coexisted with the tumour in the cases related above.

Unfortunately, a careful dissection of the blood-vessels of

the lung was not made in all the four cases, but in two of the cases (Case 2 and the case recorded by Dr. Sims) the pulmonary arteries and veins were traced.

In one of these cases (Case 2) the pulmonary artery was much contracted, but the pulmonary veins were free. In the other case the pulmonary artery, though involved in the tumour, was dilated rather than contracted, and one of the pulmonary veins was involved and lost in the tumour.

The condition of the blood-vessels was reversed in the two cases, but the condition of the lung, as regards the points to which I have called attention, was the same.

The inference from these circumstances that appears most probable is, that the thickening and adhesion of the pleura, the suppurative inflammation of the pulmonary tissue, and, in one of the cases, the inflammation of the pericardium, resulted from the tumour involving and destroying all or great part of the nerves with which these several tissues are furnished. In one of the cases (Case 2) the right pneumogastric nerve, where it forms the posterior pulmonary plexus, was traced into the centre of the tumour; and in all the other cases the situation of the tumour was such that it must have embraced this plexus of nerves, and in some of the cases the anterior pulmonary plexus as well.

It is the property of cancer to invade and destroy all tissues within its reach; and when a nervous thread passes through the centre of a hard and compact cancerous mass, its continuity must be there severed as completely as if it had been divided with the knife.

More than a century ago it was remarked—first by Parfour du Petit—that division of the sympathetic nerve in the neck causes inflammation of the conjunctiva and contraction of the pupil of the same side. Dr. J. Reid showed, in 1839, that in dogs the same influence on the eye is exerted by destruction of the superior cervical ganglion.

In 1823-4 it was ascertained, by the experiments of Fodera, Mayo, and Majendie, that division of the fifth nerve within the skull causes inflammatory destruction of

the eyeball; and soon afterwards cases were published in which similar destruction of the eye had resulted from disease involving and destroying the fifth nerve in the same situation; but, as far as I know, no corresponding results have hitherto been traced to destruction of the nerves in other parts of the body. The probable reason of this is that, from the frequent anastomosis of the nutritive nerves, it is difficult entirely to cut off from most organs the nervous influence which they serve to transmit. The lung resembles the eyeball in this, that all the nerves which supply it are comprised at its root in a very small space. The pulmonary nerves cannot well be divided in direct experiments, but they are almost necessarily destroyed by a large and compact cancerous tumour, which involves the root of the lung and surrounds the bronchus.

Another instance of inflammatory disease, probably caused in the same way, is occasionally met with in the gall-bladder, in cases in which a cancerous tumour exists in the portal notch of the liver.

I could cite from my own experience and that of others several cases of cancer of the liver, in which suppurative and destructive inflammation of the lining membrane of the gall-bladder existed, and in which there could be little doubt that the diseased condition of the gall-bladder was caused in some way or other by the cancer.

The nerves of the liver are comprised in two sets: one set, derived from the solar plexus, enters the liver at the portal notch; the other set, consisting chiefly of branches of the phrenic, passes into the liver along the hepatic veins.

The nerves of the inner coats of the gall-bladder appear to be all derived from the solar plexus, and pass from the portal notch to the gall-bladder in threads, accompanying the cystic duct, so that a small cancerous tumour there situated might, by destroying the continuity of these threads, deprive the inner coats of the gall-bladder of all nervous influence.

Whether the suppurative and destructive inflammation of

the gall-bladder, in the cases I have referred to, was caused or not by loss of such influence, must, in the absence of more minute details than are recorded respecting them, be a matter of doubt; but the cases are so interesting, in relation to the effects of cancerous tumours involving the root of the lung, that I shall perhaps need no apology for cursorily referring to them in this place.

There can be no doubt that the inflammatory destruction of the eyeball from disease involving the fifth nerve within the orbit is not a solitary fact in pathology; and that, on investigation specially directed to this point, other analogous instances will be found.

P.S.—Primary cancer involving the root of the lung is a rare disease. The three cases related above are the only instances of the kind that have fallen under my observation in King's College Hospital since it was first opened for the reception of patients in 1840. The disease is, however, so peculiar in its effects, that, as was shown by Dr. Stokes in an admirable paper on this subject published in the 'Dublin Journal of Medical Science' for 1842, a diagnosis of it may sometimes be made with much confidence. The elements of diagnosis consist—

1. In the signs that give evidence of the existence of a tumour.

2. In the absence of strong pulsation and of the morbid "bruits" that usually attend aneurismal tumours.

3. In the occurrence of hæmoptysis and other signs showing that the lung is involved in the disease.

4. In a distended and varicose condition of the superficial veins of the chest.

(A cancerous tumour usually affects the venous circulation and the nutrition of the lung more than an aneurismal tumour of the same size, because it grows into and blocks up the veins, and converts into cancer the other tissues which it embraces in its growth.)

5. When the disease occurs in a person under thirty, the age affords an additional presumption that the tumour is not an aneurism.

TWO CASES

OF

EMPYEMA,

ILLUSTRATING

THE ADVANTAGE OF MAKING TWO OPENINGS, AND ADOPTING THE PLAN OF "DRAINAGE,"

IN THE

OPERATION OF PARACENTESIS

IN THAT DISEASE.

BY

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PHYSICIAN TO THE MIDDLESEX HOSPITAL.

FOLLOWED BY SOME REMARKS ON THE OPERATION AND ON THE PLAN OF TREATMENT BY "DRAINAGE" GENERALLY.

By CAMPBELL DE MORGAN, F.R.C.S., Surgeon to the Hospital.

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Every hospital physician must have met with many cases of empyema, and many also are recorded, in which it was found impossible, after repeated operations by one opening, to prevent the accumulation of matter in the pleural cavity. Even in cases where two openings have spontaneously occurred there has been great difficulty in securing a free exit for the fluid. It is unnecessary to point out the inconvenience of this state of things. The

matter being retained in the pleural cavity and exposed to the air, soon undergoes decomposition, to the serious detriment of the patient's health and comfort. Besides the irritative fever, exhausting sweatings, and occasional attacks of diarrhœa, the fetor of the discharge becomes insupportable to himself and to those about him. Where only one opening is present the pleural cavity may be likened to a barrel without a counter-vent, and the escape of the fluid must be irregular and only partial. The plan of "drainage," first suggested and practised by Chassaignac for the healing of sinuses, would seem, from the success attending its adoption in the two following cases, to be calculated to obviate effectually these inconveniences, and to place patients so affected in a position, by which the usual process of recovery may be greatly promoted. Chassaignac's plan is very simple. An india-rubber tube, having a diameter of about the sixth of an inch, and perforated at frequent intervals by notching with scissors, is passed through the abscess or sinus, and the matter continuously and uninterruptedly exudes through the perforations. It requires no very great foresight to see that this plan by "drainage" will be found to be applicable, not only for the purpose of draining the pleural sac in cases of empyema, but for the radical cure by obliteration, under conditions otherwise favorable, of ovarian sacs, hepatic abscesses, and so on. In the first case that I shall bring under the notice of the Society it will be clearly seen that there was no probability of getting rid of the fetor, and placing the patient in a state favorable to the healing process, except by the adoption of some such plan as that of Chassaignac; and that if it had not been speedily adopted, the patient's existence, rendered miserable by so many years of suffering in many ways, must soon have been cut short. But, however clear was the propriety, nay, the necessity, of its adoption in the first case, it was only after long consideration that I ventured to have recourse to it in the second case, and to recommend to the profession this mode of treatment. With reference to the mode of performing the operation, I have nothing to add to

the description which Mr. De Morgan has given as an appendix to this paper, except that I think it would be better *in all cases* of *paracentesis thoracis* to make a small opening with a scalpel previously to introducing the trocar and canula. The pressure required for the perforation of the chest-wall by a trocar is frequently so great as to occasion much pain and distress, and sometimes it happens that the edge of the canula, in its transit through the intercostal space, gets hitched in a portion of muscle, tendon, or integument, and considerable force, and frequent attempts to introduce the trocar, may be rendered necessary. With respect also to the administration of chloroform, I think its use is contraindicated on many grounds. In the first place the operation of paracentesis is not, or ought not to be, a very painful one; secondly, no one will say that the administration of chloroform does not give a severe shock to the system. The feeling of illness and the sickness that so frequently follow the administration of this drug had much better be avoided in such persons as are the subjects of empyema, especially under circumstances requiring the operation of paracentesis. The administration of chloroform undoubtedly exerts a more or less paralysing influence on the heart, and indirectly, if not directly, a retarding influence upon the circulation of the blood through the lungs. These effects, independently of the shock, are certainly not desirable in a patient labouring under empyema, who, in nearly every case, has only one lung to breathe with, and in whom the fluid is to be withdrawn from the pleural cavity, and more or fewer important changes in the relative situation of the important organs of the chest must necessarily take place.

The history of the first case is as follows:

William Pye, æt. 17, was admitted into the Middlesex Hospital in April of last year. Up to the age of twelve years this boy had always enjoyed good health, and had lived in a healthy part of the country. Two sisters, however, aged respectively eighteen and twenty years, died of consumption;

and his mother is represented to be delicate and subject to a cough. The father is healthy.

About the age of twelve years he had an attack of measles, from the effects of which he continued to suffer for some weeks, when he was seized with what he calls "inflammation of the lungs." From the account which he gives of himself, it would seem that this attack terminated in abscess, for soon after its commencement he vomited (as he says) a good deal of matter. He states that the vomiting occurred in this way,—that during a paroxysm of cough he brought up a considerable quantity of "matter," that this made him retch violently, and that the effort of vomiting apparently had the effect of making him expectorate a still greater quantity of the same kind of matter. In the course of a short time, the purulent expectoration being still present, it was found that effusion had taken place in the right pleura, for the removal of which a succession of setons was had recourse to. But the setons soon healed, and no good effect seems to have been derived from their use. Shortly after the last seton had healed, a "pointing" was observed in the situation of one of the old setons, between the fourth and fifth ribs, about an inch and a half to the sternal aspect of the right nipple. A spontaneous opening speedily followed this "pointing," and a large quantity of purulent and highly offensive matter is reported to have been discharged. Coincidently with this discharge through the opening, the expectoration by the mouth became less; and throughout his long illness, extending over a period of more than four years, he seems to be under the impression that there was some relation between the expectoration by the mouth and the quantity discharged through the opening in the chest-wall; for when that discharge was diminished from any cause, the amount of expectoration by the mouth was invariably increased, and *vice versâ*. The opening continued to discharge this fetid pus up to the 5th of January last, when a counter-opening was made, and Chassaignac's tube inserted.

About two years after the spontaneous opening occurred, when about fourteen years of age, he was admitted into St.

Bartholomew's Hospital, from which institution he was discharged in about a month, without any perceptible change having taken place in the chest.

From the time of his admission in April, 1858, to the following July, when he came under my care, a supporting plan of treatment had been pursued, and his general health had somewhat improved. The local complaint, however, remained much the same. When he first came under my notice, the following is briefly the state which he presented: There was considerable emaciation, occasional hectic flushings, the legs and feet were œdematous, the superficial veins on the right side of the thorax and abdomen were much enlarged, appetite indifferent, difficulty in lying in the recumbent position, frequent cough with copious puriform expectoration. The affected side was somewhat flattened, and measured about an inch less than the opposite side; the opening red and irritable, and discharging freely a highly offensive purulent matter. There was no albumen in the urine, and, so far as one could judge, that excretion was normal, save that it deposited a considerable quantity of the lithates and purpurates.

On the right side, at the extreme apex, the percussion resonance was dull. Below this, down to a little below the opening, there was a tympanitic sound elicited on percussion, and below this a perfectly dull sound, which was succeeded by a tympanitic sound on making him lie on his back. At the apex a faint blowing murmur was detected by auscultation, accompanying inspiration and expiration. Below this there was amphoric respiration, voice, and cough. On the opposite, and apparently sound side, the percussion resonance was uniform and preternaturally clear, and the respiration was puerile. He continued to take quinine and iron, and cod oil, with palliatives for the cough, and occasionally aperients and diuretics. At the same time he was placed on full diet, with wine and beer. Under this treatment his general health was somewhat improved, and the œdema in the legs was diminished, but not removed. On the 27th of September, five months after his admission into

the hospital, he had a sharp attack of scarlatina. This gave rise to serious embarrassment in many ways, and placed his life in imminent peril, not only in taxing his small amount of strength, but in producing an erythematous condition of the integuments of the lower extremities. He continued, however, to take quinine and iron, according to the following formula, every six hours:

℞ Quinæ Disulph., gr. j;
Potassæ Chloratis, gr. v;
Acidi Nitro-hydrochlorici, ♏iij;
Tinct. Ferri Sequichlor., ♏x;
Aquæ ad ℥iss.

Water dressing was applied to the erythematous parts.

In the course of a fortnight he completely recovered from all the consequences of the fever, and was much in the same condition as before the attack. The discharge from the wound was so offensive, in spite of the daily introduction of a weak solution of Condy's fluid, that loud complaints were made by the other patients, and serious inconvenience was apprehended in this respect. Under these circumstances I determined on making a counter-opening in the chest-wall, as far down in the pleural cavity as it could with safety be made, and introducing the drainage-tube. With this view I consulted my colleague, Mr. De Morgan, who acquiesced in the propriety of the measure, and the operation was at once performed by him, whilst the boy was under the influence of chloroform. The manner in which the operation was performed, Mr. De Morgan has been so good as to describe, and will be found at the end of this communication. The success of the experiment exceeded our most sanguine expectations. The discharge from the opening, from being thin, unhealthy, and intolerably stinking, became much reduced in quantity, thick, and nearly free from odour; his general health rapidly improved, the œdema of the legs quickly diminished, and he was soon able to sit up for several hours every day, which he had not been able to do for the past five years, during the whole of which time he had been almost constantly confined

to bed. The discharge now (April 3d, three months after the operation) scarcely amounts to two or three drachms in the twenty-four hours, and he is able to walk in the garden with the assistance of a stick, and will in a few days be discharged.

With reference to his requiring the assistance of a stick when walking, it should be stated that this assistance is required solely from the weakness of his legs and tenderness of his feet, from the length of time that he has lain in bed. It is very probable that during this long time of desuetude, about five years, the muscles may have undergone some degeneration. So far as his respiration is concerned, there is no impediment to his taking a moderate share of exercise.

The next case is that of Henry Thompson. He was sent to the hospital by Dr. Watson, who had seen him on several occasions prior to his admission. The following is the account of his case, taken at the time of his admission, August 13th, 1858:—Aged 24, a coachmaker; height 5 feet 10 inches; fair complexion; muscular system well developed. Father died at the age of twenty-four, of phthisis. An only child. Always had good health up to two or two and a half years ago, and considered himself strong and robust. Had measles when five or six years old, from which he recovered completely, and never had a cough prior to the period above stated. About this time he first began to be more or less troubled with a dry, hacking cough, which continued to increase slowly until he was induced to apply for relief to the Consumption Hospital in the City Road. About two months after he had been under treatment he was attacked with hæmoptysis, during which he lost, for the first six days, on the whole, about a quart of blood. In November last, nine months ago, he was attacked with the usual symptoms of pleurisy, leaving a copious effusion in the left pleural cavity, which has continued there to the present time. Last November, he consulted Dr. Watson, whose report is as follows: "In his right lung the respi-

ration was loud, puerile; in front of the left side no respiration could be heard, but the sound on percussion was unduly resonant; between the scapula and spine there was amphoric breathing, and he told me that he had had a splashing in the side on sudden jars of the body. I could produce no splash then on succussion. I concluded that he had pneumothorax, and that a communication had been made between the lung and the cavity of the pleura in the progress of tubercular disease. By the 23d of December the amphoric sound had disappeared; he had grown fatter; there was evidence of an accumulation of fluid in the left pleura. I saw him no more till two days ago. His left chest is now distended with fluid, and the heart pushed over to the right."

On his admission he had the appearance of good health; bony frame large; muscular development considerable, with a fair amount of subcutaneous fat. Expression of countenance free from distress. Breathing easy; number of respirations 18 in a minute. Cough seldom troublesome, and brought on only by any unusual exertion; is not distressed with it at night, nor has he any nocturnal sweats. Decubitus equally easy in all postures. Bowels regular of late, but previous to illness habitually costive. Has no griping pain in the bowels, and has not at any time been subject to diarrhœa.

Physical signs.—Chest (left side): A tape placed horizontally round the chest, an inch and a half above the xiphoid cartilage, shows that the left side measures one inch more than the right. On a level with the apex of the axillary region the tape indicates scarcely any difference between the two sides. Amount of mobility of the left side, on a full inspiration, a quarter of an inch; that of the right, three quarters of an inch. Dulness and increased fulness extend down to the level of a line drawn from the umbilicus. Palpation detects a very slight vocal fremitus in the infra-scapular fossa and in the left inter-scapular space. It is absent in the lower regions of this (the left) side. On the right side, vocal fremitus and resonance well

marked. Some vocal resonance is heard where the vocal fremitus is felt on the *left* side. This resonance has but a very slight ægophonic character. There is a faint respiratory murmur in the upper posterior and anterior regions of this side. It is absent in the other regions.

Right side: Apex down to second rib dull on percussion. The respiratory murmur harsh, and the expiratory murmur as long as, and in some points longer than, the inspiratory. The other regions on this side are unusually resonant, and the respiratory murmur supplementary, but otherwise healthy.

Heart: Strongest impulse felt about two inches below, and about an inch to the external aspect of, the right nipple. The sounds muffled or distant. No abnormal *bruit*. Tongue natural. Appetite good. Pulse 84.

Here, then, was indubitable evidence of effusion in the left pleural cavity to a very considerable extent. There was evidence, also, especially when taken in connexion with his family and his own previous history, of tubercular consolidation in the apex of the opposite lung. The question naturally arose whether, taking into consideration the apparently good state of his general health, the freedom from distress of any kind, and the strong evidence of the presence of tubercular consolidation of the right apex and of the tubercular origin of the empyema, it was justifiable to have recourse to the operation of paracentesis thoracis. I confess that I felt unwilling to undertake the responsibility of recommending it—certainly not until the usual remedies for producing the absorption of the fluid, however small the probability of their producing this result appeared, had been fairly tried, or some symptoms of distress from the large amount of fluid began to show themselves. There was but small chance of the lung expanding on the withdrawal of the fluid, even if it had been sound, which it is almost certain it was not. The lung had been compressed by fluid for more than a year; it was probably bound down by the usual thick false membrane which forms under these circumstances, except at the

apex, where it was held to the opposite chest-wall by old adhesions. He remained in the hospital until the following December (four months), when he was discharged at his own request, and made an out-patient. The fluid was seemingly much diminished. The greatest impulse of the heart was felt about an *inch* to the *mesial* aspect of the right nipple, instead of, as on his admission, at the same distance on the *external* or distal aspect.

On the 18th of January, however, he was again admitted. The breathing was now much oppressed, and his general health was apparently suffering from the increase of the effusion. The heart had now returned to its old situation, an inch or more to the right of the nipple. From the apparent success in Pye's case, I determined on having recourse to the operation of paracentesis by a double opening and by "drainage." Mr. De Morgan was again kind enough to give me his valuable assistance; and on the 21st of January, three days after his readmission, Mr. De Morgan made an opening between the fifth and sixth ribs by means of a trocar. There was some difficulty in pushing the trocar through the exceedingly thick false membrane lining the ribs. In the canula a small plug was introduced, so as to produce a more gradual escape of the contained fluid, its sudden withdrawal being likely, it was feared, to bring on syncope and other untoward effects. In the course of the next twenty-four hours eight quarts and half a pint of unhealthy-looking sero-purulent matter had escaped, and the opening was still freely discharging. Four days after the first opening the second opening was made, and the tube introduced. After about ten days, however, it was found that the second opening was not made sufficiently low—the matter remained so long in the chest that it became decomposed and extremely offensive. A second counter-opening was then made as low down as the thick probe, mentioned in Mr. De Morgan's account, could be carried, and the opening made as described. After this the pus soon lost its offensive odour, it rapidly diminished in quantity, and the man's health so rapidly improved, that

he was able to get up in a few days. This, I think, shows what I was convinced of from the first—that the counter-opening should always be made as far down in the pleural cavity as possible, even to the extreme apex of the triangular space formed by the attachment of the diaphragm to the thoracic wall. The complete success of the operation will much depend upon this recommendation being strictly observed.

In the interval between the first and second counter-openings some black, solid matters made their escape with the pus. On examination by the microscope, these matters were found to consist of altered pigment-cells, crystals of hæmatin, crystals of cholesterine, and a considerable quantity of yellow fibrous tissue. None of the elements of tubercle could be detected, nor any of the other elements of lung-tissue. Supposing that these matters were the remains of some of the contents of the cavity in the lung, which had found their way into the pleural sac at the time of the occurrence of the pneumothorax, it was not to be expected, after the lapse of so long a time, that these destructible tissues should have remained undecomposed, and in a state to be recognised on examination.

For a few days after the first operation he of course felt weak, but not at all more so than was to be expected. He was also troubled with some cough. But what gave us the most apprehension was a jaundiced condition that made its appearance about five or six days after the operation. The skin was deeply tinged, the urine was very yellow, and indicated, by the usual reagents, the presence of a considerable admixture of bile. There was frequent vomiting; the pulse was very quick and weak; the appetite indifferent; and there were occasional profuse sweatings. Calomel and saline aperients, followed by vegetable tonics and the mixed acids, succeeded, however, in removing this condition. But it returned again about six weeks afterwards. How far this jaundice may be ascribed to the altered position of the parts within the chest and abdomen, consequent upon the withdrawal of the fluid, it is impossible to say. Probably, it

was in great measure due to the pressure, so long continued, of so much effusion upon the inferior cava, and the abnormal position and altered relative situation of the heart and this vessel, leading to congestion of the liver. It was found necessary during the treatment *before the operation* to give remedies calculated to relieve the liver and the portal system.

It is not the object of this communication to give every minute symptom that presented itself after the operations, or the medicines prescribed for its removal. It would needlessly occupy the time of the Society. It is sufficient to say, that after the removal of the jaundice on each occasion that it presented itself he gradually improved in health and strength, and was able to return to his home on the 5th of April, between two and three months after the first operation.

In cases of empyema, from whatever cause induced, where there is good ground for concluding that the lung is inexpansible, I think few will be prepared to deny the advantage of making two openings in the operation of paracentesis thoracis, and applying the plan of "drainage." The only probable exception to this is where empyema occurs in a young child, in whom the restorative powers are strong, and the chest-wall is so elastic that it readily falls in and adapts itself to the diminished quantity of fluid within the chest.

In volume ix, second series, of the Society's 'Transactions,' there is a case of empyema reported by Dr. Theophilus Thompson, in which, if this mode of procedure had been adopted, there would have been no necessity for so many operations of tapping, and the recovery would have been greatly promoted. There are some cases, also, reported by Dr. Hamilton Roe, in his interesting and valuable paper on Paracentesis Thoracis, in the same volume of the 'Transactions,' where the plan by "drainage" would evidently have saved the patient much suffering, and the medical attendants considerable anxiety and trouble.

How far the plan is applicable for the removal of serous effusions there will be greater difference of opinion. For my own part, I should not think of having recourse to it in those cases. In such cases, as little air should be allowed to get access to the pleural sac as possible, and in the majority of cases the fluid that is not removed by the canula will soon be taken up by the absorbents, and a recovery produced without such an operation as I have recommended in empyema.

APPENDIX TO DR. GOODFELLOW'S PAPER ON THE TREATMENT BY DRAINAGE;

By CAMPBELL DE MORGAN,
SURGEON TO THE MIDDLESEX HOSPITAL.

The treatment of deep-seated and extensive collections of matter by "drainage," first adopted by Chassaignac, has been largely and in many cases very beneficially put in practice in the Middlesex Hospital. The operation consists in the passing through the abscess, at two points, of a fine india-rubber tube, perforated at small intervals. The ends of the tube which project from the opposite sides of the abscess are then tied together, and the matter is allowed to drain away and to discharge itself through the perforations made in the tube.

The advantages of this proceeding are in many cases obvious. The difficulty which is always experienced in keeping free the openings leading to deep chronic abscesses is overcome, and the matter is discharged as soon as formed. Where simple openings are made into such abscesses, there is always a tendency to their more or less complete closure, and the frequent introduction of a probe is rendered necessary. This implies that the abscess is not kept continuously

empty, but that a certain amount of matter will lodge in it until the opening be freed. If two openings be made the matter will find its way by the one which offers the greatest facilities for its exit, and the other will close entirely without constant care. It seldom happens that the matter in a chronic abscess is discharged so continuously but that a considerable quantity can be made to flow out when pressure is applied, and hence the conditions under which an abscess will most readily heal are absent. I have now under my care in the hospital a man whose foot I removed by Syme's operation. The parts did not take on a healthy action, and matter formed and lodged between the faces of the stump. Counter-openings were made, but the pus never discharged itself completely; a quantity could always be forced out on pressure. A drainage-tube was introduced from the front of the stump to the heel. In the course of a day or two the fulness and œdema of the stump subsided, no matter collected, and not more than a couple of drachms was formed in the course of twenty-four hours. If the tube was removed, the old state of parts speedily returned. Not the slightest irritation was produced by the tube, although it was worn for a considerable time.

There is no situation in the body to which these remarks apply so forcibly as the thorax. This cavity, when filled with pus, while the lung, as is usually the case, is bound down and inexpansible, is in much the same state as a barrel filled with fluid. If one opening be made in it the fluid will be forced out in the first instance by the pressure of the distended sides, and no more can pass out unless air be admitted to supply its place. So soon as the canula is withdrawn the matter re-collects, and a partial discharge of it only takes place. When the operation has been performed in the usual situation there will always be a considerable accumulation of matter in the chest below the level of the opening. Another evil attending this continuance in the cavity of a quantity of matter which cannot find exit as soon as formed, and is at the same time exposed to air, is that it soon becomes decomposed, and by its disgusting

smell adds much to the patient's trouble. If a simple counter-opening be made, it will for the moment check or diminish this, but very speedily one or other orifice will close, and it will be found impossible, without the introduction of a tube, to keep them both open in such a way as to admit air freely on one side, while the matter can drain away as soon as formed on the other. These are essential conditions to keeping the chest free from matter, and the pus itself from being decomposed.

The introduction of a "drainage" tube fulfils completely these necessary conditions. The openings are always free; the matter is discharged drop by drop as it forms, so that if the tube be suitably placed there is never any collection whatever of pus in the thorax; no time is given for decomposition, and the pus is therefore discharged in a healthy and pure state.

The tube does not appear to give rise to the slightest irritation or pain, and the patients can walk and move about without the consciousness of its presence, and can lie comfortably in any position.

The operation is a very simple one. A puncture with a trocar, or a simple incision, may be made into the cavity of the chest at the usual place, between the fifth and sixth or sixth and seventh ribs, or indeed in any convenient situation. A firm, long, iron probe, somewhat bent, is then passed through the opening and directed towards the lower and back part of the cavity—the lower the better. If the end of the probe be made to press against the sides of the thoracic walls, it can be felt from the outside through the intercostal spaces, though perhaps obscurely, owing to the thickness and toughness of the false membrane within. The lowest and most appropriate site in which the probe can be felt having been selected, an incision is made upon the end of the probe, which is then brought through the opening thus made. A strong piece of silk thread is passed into the eye of the probe and drawn through the two openings, and the drainage-tube, being firmly tied to one end, is then drawn

through by means of the silk. The ends of the tube are tied together, and the operation is completed.

The only reason for using a silk thread wherewith to draw the tube through the openings, is that a smaller opening will answer. The large end of a strong iron probe, threaded with an ordinary-sized drainage-tube, would require for its passage a larger opening than is wanted.

AN INQUIRY INTO THE NATURE OF THOSE CASES

OF

STRANGULATED OBLIQUE INGUINAL HERNIA,

TERMED

"RÉDUCTION EN BLOC, OU EN MASSE;"

WITH SPECIAL RELATION TO THE

ANATOMY OF THE ACTUAL LESION,

AND PRACTICAL DEDUCTIONS DERIVED FROM AN EXAMINATION OF THE CASES.

BY

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"Il ne faut jamais travailler dans le but de soutenir une théorie, parce qu' alors l'esprit se prévient."

"Notre seul but doit être la découverte de la vérité." — CUVIER ('Opinion sur les théories').

MODERN writers on the subject of strangulated hernia have recognised the occurrence of cases, in which the hernial tumour has been pushed into the abdominal cavity by the forcible efforts of the patient, or of the surgeon.

This mode of reducing a hernia has received the name of "réduction en bloc," or "en masse."

The perplexing nature of these cases, their urgency, the fatal results attending them if they are not at once recognised, and the impediment to the reduction of the hernia immediately removed, may plead as an excuse for intruding the following observations on the members of this Society.

From the time of Le Dran to the present day, the practicability of returning a hernial protrusion into the abdominal cavity, *together with its investing sac*, whilst constricted or strangulated by the orifice of the sac, has been admitted. This doctrine is now indiscriminately applied to inguinal, inguino-scrotal, and femoral herniæ. The principle was first, however, enunciated by Le Dran in consequence of having met with a case of strangulated *femoral* hernia, in which, although the tumour had been reduced, the patient died with all the symptoms of strangulated bowel persistent. *Post-mortem* examination demonstrated that the orifice of the hernial sac strangulated the bowel, and that *the sac with its contents* had been pushed within the abdominal walls. ('Obs. de Chir.,' 2 vols. 12mo., Paris, 1731.)

The purport of this inquiry is to ascertain—

1st. The applicability of the terms "réduction en bloc," or "en masse," to cases of *inguinal* and *inguino-scrotal* herniæ *exclusively*; whether the lesion stated to take place does ever happen; in point of fact, is it practicable to detach the hernial sac from its connexions in the scrotum, push it through the abdominal rings and inguinal canal, and lodge it within the abdominal walls?

2dly. To endeavour to demonstrate the actual morbid anatomy of the lesion, by giving a description of the dissection of cases, in which this accident is said to have occurred.

3dly. To show in which varieties of inguino-scrotal hernia this accident most frequently happens.

4thly. To point out the practical inferences deducible from the examination of these cases.

PART I.

After a critical examination of the cases on record termed "réduction *en masse*," we may arrange them in two classes.

CLASS I.—The cases in which the patient died with the strangulated bowel unrelieved.

CLASS II.—The cases in which a cutting operation was performed for the purpose of relieving the strangulated bowel.

CLASS I.—Upon reference to the cases recorded by MM. Saviard,[1] Ricot,[2] Arnaud,[3] Scarpa,[4] Sir Charles Bell,[5] M. Laugier,[6] and some others, the nature of the lesion described may be classified under three heads.

1. Those in which *the hernia* is pushed out of sight, and is found, *post mortem*, between the peritoneum and the abdominal walls.

2. Those in which *the hernia* is supposed to be reduced, but is found, *post mortem*, to be *in a pouch* within the abdomen.

3. Those in which *the orifice of the hernial sac has been torn off*, which circumstance permits the hernia to be pushed within the abdominal walls, but not into the peritoneal cavity.

In some of the cases the hernial sac is mentioned, but I cannot find an instance in which the description might lead to the belief, that the sac had been detached from the scrotum; and in the case described by Sir C. Bell, the illustrative woodcut shows clearly that no such lesion could have taken place.

[1] 'Obs. de Chir.,' Paris, 1702.
[2] Arnaud, 'Diss.,' &c., p. 382, obs. vi.
[3] Op. cit., p. 389.
[4] 'Sull. 'ernie,' 2d edit., p. 49.
[5] 'The Lond. Med. Gaz.,' vol. i, p. 485.
[6] 'Bull. de l'Acad. Roy. de Méd.,' Juin, 1840.

Class II.—Let us now examine the nature of the operation performed for the relief of the strangulated bowel, and especially as regards the situation, or region, in which the hernial sac was opened. Be it remembered, that it is an essential characteristic of this variety of hernia that *the sac* has been pushed into the abdominal cavity, and must, therefore, in the event of the protrusion having descended into the scrotum, have been detached from its scrotal connexions.

I would ask, is it possible that so much injury could be inflicted on the tissues of the scrotum without some very marked indications of its existence?

In a majority of instances, the hernia seems to have descended into a scrotal sac of larger or smaller size, yet it cannot escape observation that there is not, in one of these cases, a single remark made upon the state of the scrotum. Would not this region have exhibited signs of contusion? Would not ecchymosis have been developed, or, after a few hours, inflammatory effusion poured into the scrotum? Indeed, in many cases several hours elapsed between the supposed reduction of the sac with its contents and the operation, and yet the condition of the scrotum is not alluded to in a single instance.

The only cases in which the slightest allusion is made to any injury which might be presumed to result from the forcible tearing asunder the connexions between the hernial sac and its investing textures are related by Mr. Luke and Mr. Curling.

Mr. Luke writes—"A local indication had also now arisen by the formation of a tumefaction along the course of the inguinal canal." This was on *the eighth day* after the commencement of the attack. Continuing the narrative, "an incision was made over the seat of tumefaction, from which a quantity of highly offensive, sanious fluid exuded, and by the infiltration of which into the cellular texture of the part the tumefaction had been caused." ('Med.-Chir. Trans.,' xxvi, p. 165.)

Mr. Curling writes—"An incision having been made

into the spermatic cord, it was found to be swollen, chiefly from œdematous effusion." ('Lancet,' 1850, vol. ii, p. 81.)

But one inference can be drawn from the omission of all mention of any morbid condition of the scrotum in the cases, in which this injury is supposed to have been inflicted.

But, assuming that a morbid condition of the scrotum and inguinal canal did exist, and that, through neglecting to observe it, the circumstance had not been stated, we are relieved from all doubt upon the matter by Mr. Luke, who regards the absence of all such indications of injury as one of the most prominent features by which to form a diagnostication of the accident. He writes, when describing the operation, and after stating that the tendon of the external oblique muscle, the abdominal ring, and spermatic cord were exposed, "a finger was then introduced into the ring to feel for some indication of the presence of a tumour within the inguinal canal, but there was not any discovered. The canal was next laid open by a division of the tendon of the external oblique muscle. It was observed that the spermatic cord *was clear and unobscured* by any superjacent structure, except by a small lobule of fat which overlaid its upper part. While pursuing the examination towards the internal ring, some cellular membrane lying on the inner aspect of the cord, appeared more condensed than usual in that situation; which, upon closer investigation, proved to be a condensed capsule, containing an empty cavity within it, sufficiently large to contain a small egg. It was thought probable that this capsule had formed an investment to the hernial sac, and was now left empty by the reason of the reduction, that at this time was strongly suspected to have been effected." ('Med.-Chir. Trans.,' xxvi, p. 170.) Surely, if this had taken place, the "capsule" would have afforded some, if but slight, evidence of the laceration?

In another paragraph of the same paper Mr. Luke writes, (p. 175)—"The presence of sac, even without hernial contents, causes an abnormal fulness in the part, easily ascertainable by examination. The absence of such fulness in a part, when hernia is known to have previously

descended, necessarily leads to the conclusion that the sac upon which it depended, has been *displaced*, and probably returned together with the hernia."

An explanation of the circumstances might be brought forward by stating that the sac is not supposed to be wholly and entirely returned into the abdomen, but only a portion of it, and that in some cases but a partial reduction is effected, leaving the lowermost part of the body and fundus of the sac behind. But I contend that, if any part of the sac be detached from its scrotal connexions, some traces, however small, would remain to indicate the laceration.

Again, admitting that the hernial sac had been detached and pushed into the abdomen, and that it had been again brought down before the operation, or at the time of its performance, would not the operator find its body and fundus lying loose in the inguinal canal or scrotum, and but slightly attached at its neck? But in the relation of the cases such an observation does not occur, and, with the exception of a few instances, the hernial sac has been always opened before the sac enclosing the hernia has been reached. Nay, further, the operator, in many instances, states particularly that he experienced the greatest difficulty in bringing down the sac, when he had at last reached it. Does not this statement militate against the fact of the sac having been detached from its connexions? But we may be told that it has formed new adhesions in its novel position. Has there been time for these to be developed?

From such statements as the above, we may conclude that surgeons have not observed any appearances, either before the operation or at the time of its performance, leading to the inference that the hernial sac had been torn from its connexions in the scrotum and inguinal canal; but that, because the hernial sac was not clearly seen, it has been assumed that it was pushed into the abdomen. Mr. Luke is perfectly correct in stating that the sac is "displaced," but I hope to be able to demonstrate that it is not pushed bodily into the abdomen by being torn away from its connexions in the scrotum and inguinal canal.

Having examined the nature of the operation performed in these cases, and stated that in the majority of instances the hernial sac was opened in the inguinal canal, we may take advantage of the fatal cases to ascertain the local injury resulting from the supposed reduction of the hernial sac with its contents into the abdomen.

I have collected the record of eight cases, in which it is said that the hernial sac was found in the abdomen and opened within its cavity, or pulled out for the purpose of liberating the strangulated bowel. Of these cases only one terminated fatally, and this fatal result is entirely attributable to the long period the bowel had been strangulated before the patient came under the treatment of the operator. That period was six days. In the relation of the case ('Lancet,' 1850, vol. ii, p. 81) it is stated, that the inguinal canal was exposed, but no hernial sac could be made out. An empty sac was found at the back of the cord. Something like a tense sac was felt in the abdomen at the situation of the internal ring. "An opening was next made into what seemed the peritoneum, and intestine escaped." From this sac a small aperture was felt, which was divided. *Necropsy.*—General peritonitis. "In the right iliac fossa, between the peritoneum and fascia transversalis, there was a large hernial sac, which contained a coil, about five inches, of intestine . . . . The neck of the sac appeared to correspond with the internal ring, but to have become displaced . . . and the dilated pouch of peritoneum which formed the sac, instead of descending along the inguinal canal, had passed into the iliac fossa, and remained within the boundary of the abdominal walls."

In the case related by Mr. Luke, to which allusion has been made before, he states—"The large hernial sac discovered during the operation was brought more fully into view," (this sac having been incised by the operator in the inguinal canal.) "It was found to occupy a considerable space just within the abdominal parietes, in the vicinity of the internal ring. The fundus of the sac lay a little below its level towards the cavity of the pelvis, while the

neck (still contracted so as obviously to have been the original seat of stricture) lay in an upward direction towards the umbilicus, and between three and four inches distant from the situation of the internal ring." ('Med.-Chir. Trans.,' xxvi, p. 162.)

In the relation of the cases by Mr. Cock, he distinctly states that the hernial sac was opened in the inguinal canal, and in the detail of the necropsy he writes—"It was not until the omentum and some folds of bowel had been turned aside that the adventitious pouch was brought into view, and then the ring of peritoneum constituting the entrance into it was clearly seen, as depicted in the drawings." (2d case, R. E—, p. 59.) The drawings made from a dissection of the parts, show very clearly, especially Plate III, that the hernial sac still maintains its relations to the surrounding parts, and that, in the efforts to reduce the hernia, *the protrusion only, and not the sac,* was reduced within the abdominal walls, but not within the peritoneal cavity. In an appendix to this paper (p. 66) Mr. Cock relates a case, of which I made the *post-mortem* examination, and in conjunction with Mr. Cock. It was demonstrated that the intestine which had formed the hernia, had been pushed through *a lacerated opening* in the neck of the hernial sac, and was lying *outside the peritoneum* upon the fascia iliaca. ('Guy's Hospital Reports,' 1847, pp. 55, 56.)

This, so far as I can discover, is the first recorded statement of this injury, the laceration of the hernial sac, through which aperture the hernia makes its escape, to become lodged between the peritoneum and the abdominal walls. Some authors have stated, as before described, that the protrusion or hernia is pushed outside the peritoneum, but they have not accurately explained how such an accident may occur, nor has one of them spoken of this laceration of the peritoneal sac.

A very interesting and well-described case is recorded by Mr. Reid, of Canterbury, who laid open the right inguinal canal of a man, aged seventy-five years, and exposed a tumour in its upper part, in which, when opened, intestine

was found. This formed, however, but a small part of the sac; the remainder extended so far within the abdomen that it required the full length of the index-finger to reach the stricture. The man died from acute general peritonitis, and the post-mortem examination afforded the following results. "The sac was of the form of a double pouch, with a common neck and opening. A smaller pouch, about one fifth of the whole, partly within the abdomen and partly protruding through the internal ring; a second and larger pouch, placed nearly at right angles to the former, and extending from the inner side of the internal ring *between the peritoneum* and *abdominal parietes*. . . . The sac was readily drawn out from its double position, and then formed a simple bag. The outer side of the neck of the sac was only slightly separated from its connexion with the abdominal parietes." ('Prov. Med. and Surg. Journal,' 1849, vol. xiii, p. 41.)

This description of the double pouch accords precisely with the appearances in the case to be related directly; and a hasty perusal of the post-mortem examination might lead to the belief that the hernial sac was pushed into the abdomen. But what are the facts pointing to such a conclusion? No morbid condition of the scrotum or inguinal canal is stated to have been observed before the operation, or during its performance, to lead to the inference that the sac had been detached from its connexions, and in consequence of that separation pushed into the abdomen. The author terms the case one of "incomplete réduction en masse," thus intimating an impression on his mind that it did not fully accord with the circumstances of the accident. The statement that the second and larger pouch was placed *between the peritoneum and abdominal parietes*, is to be explained in two ways only. Either the hernial sac was dilated into this second pouch, and thus a duplicature of peritoneum formed; or, the neck of the hernial sac was torn, and the hernia escaped through it outside the peritoneum, and thus was lodged in a region where there seemed to be a newly formed pouch, although in reality there was not.

From the foregoing facts the following conclusions may be deduced:

1. That although the hernial sac is *displaced,* it is not detached from its scrotal envelopes.

2. That the practicability of opening the hernial sac in the inguinal canal is good evidence that it was not pushed into the abdomen.

3. That the difficulty in bringing out the sac containing the hernia from the abdomen, when it is said to be therein, is evidence that its connexions must be more firm in that region than would result from the mere pushing it there.

4. That the situation of the hernia has been pointed out, in some cases, although the exact nature of the lesion has not been fully described.

5. That the details of the cases are not in accordance with the presumed or accepted conditions of the accident.

6. That the evidence of the practicability of the patient or a surgeon reducing, into the abdomen, a *scrotal hernia,* together *with the sac* still strangulating its contents, is, at the present moment, equivocal.

7. And, therefore, that the term "réduction en bloc, or en masse" is not so applicable to cases of oblique inguino-scrotal hernia as to other species.

PART II.

The next point in this inquiry is to demonstrate the actual morbid anatomy of the lesion, and I shall briefly describe the cases which I have carefully examined. I do not pretend that the opportunity has occurred to me to observe every variety that may possibly occur, but, after the facts I shall immediately describe, future observers will have the opportunity of either corroborating or adding to those which I adduce.

The first case seems to represent only one stage of the

mechanical results arising from pressure upon a hernial tumour. In this instance the hernia, a knuckle of ileum, was strangulated by the orifice of the peritoneal or hernial sac, and so tightly as to prevent the return of it into the peritoneal cavity. The result of pressure upon the scrotal tumour seems to have been—first, to push the orifice of the hernial sac away from the internal abdominal ring; secondly, to dilate that part of the hernial sac between its orifice and the internal abdominal ring into a pouch; and, thirdly, to push the hernia out of the scrotum and inguinal canal into this pouch, where it became fixed behind the internal abdominal fascia.

The hernial sac was not detached from the inguinal canal or scrotum.

Case 1.—In November, 1834, a man was admitted into Guy's Hospital, under the late Mr. B. B. Cooper, suffering under urgent symptoms of strangulated bowel. When placed in a warm bath he became faint, and whilst in this state the taxis was employed, which proved, apparently, effectual, as the hernia was displaced from the scrotum, and seemed to enter the abdominal cavity. He even expressed himself relieved. However, after recovering from the faintness, all the symptoms of strangulation of intestine returned, and with greater severity. Not the slightest indication of a hernial protrusion could be discovered by a close examination of the inguinal region, and, suffering, as was supposed, from some internal obstruction, the man died.

Necropsy.—The results of general peritonitis were seen. A knuckle of intestine, in length about two inches, was found strangulated in a distinct sac, and it was lying between the right linea ilio-pectinea and the urinary bladder. The sac, as seen within the abdomen, seemed about equal in size to a large walnut; its aperture, which formed the stricture around the intestine, was pretty firm.[1]

A preparation of the parts concerned with the hernia is

[1] 'Guy's Hospital Reports,' 1839, vol. iv, p. 331.

preserved in the museum at Guy's, and I have carefully examined it after removal from the bottle. The attachments of the hernial sac to the upper part of the scrotal region seem to be unaffected, but the neck of the hernial sac appears to have undergone a process of dilatation, just within the internal abdominal ring, and thus to have formed a pouch or secondary sac, into which the hernia was forced from the scrotum. The constricted orifice of the hernial sac, which formed the impediment to the reduction of the hernia into the peritoneal cavity, was pushed away from its connexions with the internal abdominal ring, but *there was not*, in this case, any laceration of the hernial sac.

The cases which ensue, illustrate the further progress of events, and they are, I believe, of the kind which most commonly happens. After the continued pressure exerted upon the neck of the hernial sac has dilated it into a pouch, the tissues of which it is composed, being no longer capable of resisting the force from without, gently yield under its influence. The result is the laceration of the hernial sac at that part where it is the least strengthened by the contiguous structures, which permits the escape of any serum it may contain into the subperitoneal connective tissue, and after that the hernia, escaping likewise through the same rent, is lodged outside the peritoneum, between this serous membrane and the internal abdominal fascia, not in a well-developed pouch, but in the connective tissue of the region.

In none of the cases was the hernial sac detached from the inguinal canal or scrotum.

CASE 2.— *Open inguinal portion of the vaginal process of the peritoneum from birth; hydrocele of the tunica vaginalis injected and cured. Irreducible omental hernia, serum in hernial sac; strangulated hernia, operation, death, necropsy.*

In November, 1854, I was asked to see a little delicate boy, æt. 13, whom I found suffering with right inguino-scrotal hernia.

I learned that on the 20th he had been taking violent exercise, by jumping over forms and benches; that on the 21st he had been in bed all day, and some castor oil was administered by his friends, and the next morning he vomited. A surgeon saw him, and discovered the hernia, and the boy stated that the swelling became larger early in the morning of the 21st, which was the first time it had given him any pain or trouble. He was known to have been the subject of a hernia into the inguinal portion of the peritoneum for about a year, but, as was subsequently proved, he must have had an epiplocele for a longer time.

I found him suffering great pain from a large tumour in the right side of the scrotum, which projected forwards; the neck of the tumour being remarkably small and well defined, and terminating sharply at the external abdominal ring. As there could be no doubt of the existence of a hernia, chloroform was administered, and by gradual and continued compression of the swelling, *certainly without force*, it was reduced *in size*. The margins of the external abdominal ring became distinct, and I hoped I had returned a part of the protrusion. A portion, however, remained behind in the inguinal canal, and from the manner in which the hernia diminished in size, I had some misgivings as to its complete reduction within the peritoneal cavity.

Nausea and vomiting continued; the swelling became again large, and I returned to the patient at noon on the 24th. He had been greatly depressed by the chloroform, and was long recovering from its influence. It was now necessary to explore the tumour, and this was done whilst the patient was again under the influence of chloroform.

I found an old hernial sac in the scrotum, to which omentum was adherent, and a coil of intestine (ileum), which was deeply congested and inflamed. I passed my finger upwards along the lower border of the bowel, and felt what I supposed to be the neck of the hernial sac, which seemed sufficiently large to permit the replacement of the intestine; but, in doing this, I discovered that the protrusion passed behind the peritoneum through a rent in

the hernial sac. I then gently drew the bowel back, and, by tracing the omentum upwards, I felt the neck of the sac at a great depth, and then slightly enlarged it. Another attempt was made to reduce the protrusion, but it eluded my efforts to pass it through the orifice of the hernial sac into the peritoneal cavity, and glided behind the peritoneum into a space formed in the subperitoneal connective tissue of the iliac fossa, as was demonstrated after death.

The contents of the sac, except the adherent omentum, having disappeared, the wound was dressed as usual. Vomiting continued, exhaustion followed, and the boy died about ninety-five hours from the commencement of the attack.

Necropsy.—General peritonitis existed. By making the examination from the inguinal region I found that a portion of the bowel had never been returned into the peritoneal cavity, but was lying in the iliac fossa outside the serous membrane. The posterior wall of the peritoneal or hernial sac had been torn about half an inch below its orifice, and through this rent the finger passed on to the fascia iliaca. Looking towards the iliac fossa from within, the anterior abdominal walls being reflected, the intestinal protrusion was girt by the orifice or mouth of the hernial sac, and the hernia seemed to occupy a newly formed pouch in this fossa, where it was covered by the peritoneal membrane of the region.

The hernial sac still maintained its connexions with the tissues of the scrotum, and was not in the slightest degree displaced in that region.

The explanation of the perplexing circumstances attending this case is as follows. In the attempts to reduce the hernia, the mouth of the sac had been pushed upwards and away from its connexions with the internal abdominal ring and fascia. The posterior wall of the neck of the hernial sac had been torn, and through this rent the bowel escaped into the iliac fossa. Probably, also, the serum which the sac contained, oozed through it into the connective tissue of

the same fossa, which explains the gradually diminished size of the swelling by the pressure exerted upon it. At the operation, my finger at first passed through this lacerated opening, instead of into the peritoneal cavity, through the orifice of the neck of the sac, with greater facility than it did at the necropsy, although I discovered the orifice by tracing the omentum upwards, and returned part of the bowel through it. The two apertures could not be distinctly made out during life, and from their proximity, their depth, and the attachments of the neck of the sac being destroyed, the protruded bowel passed partly through the true orifice of the hernial sac, and partly through the abnormal opening in its posterior wall.

Case 3.—Of this case I made the examination, when demonstrator of morbid anatomy at Guy's Hospital, in 1847. The patient was admitted under the care of Mr. Cock, and the case is published in the 'Guy's Hospital Reports' (series ii, vol. v, p. 66).

The patient himself, after repeated efforts, had pushed into the abdomen the contents of a large hernial sac, which occupied the scrotum. This region had been empty, but when admitted it was occupied by an enterocele, which was reducible, and marked indications of strangulation of intestine continued. Mr. Cock operated upon the patient, divided the narrow orifice of the sac, and replaced the bowel within the peritoneal cavity. The man died of acute peritonitis, and, *post mortem*, a coil of small intestine was found in the right iliac fossa, lying outside the peritoneum upon the iliacus muscle. It had reached this situation by escaping from the hernial sac through a laceration in its neck, and although the coil which formed the original hernia had been returned by Mr. Cock at the time of the operation, another coil had descended subsequently, and now occupied this abnormal position. The secondary hernia was the consequence of violent muscular movements made by the patient after the operation. The appearance of that part of the small intestine which had recently es-

caped through the lacerated hernial sac was clearly indicative of the fact, for it had not undergone any morbid change, whilst that which had been subjected to protracted strangulation was lying in the abdomen, and its morbid condition clearly indicated the constriction which had been exerted around it.

The hernial sac was situated in the inguinal canal and the upper part of the scrotum, and there were no indications, either before the operation, during its performance, or afterwards, that its connexions with the scrotum had been torn. The hernia was developed when the patient was about eighteen or nineteen years old, and although it was not in contact with the testis, it doubtless descended into a canal dependent upon the non-closure of the inguinal portion of the vaginal process of the peritoneum.

Case 4.—The details of this case are reported by Mr. Aston Key, in the second edition of Sir Astley Cooper's work on hernia.[1]

The man was admitted under the care of the late Mr. Morgan, in June, 1825. H—, æt. 58, had been the subject of right, oblique, inguino-scrotal hernia for several years. It had been sometimes irreducible for many hours. Before admission a surgeon had endeavoured to reduce the protrusion, but he did not succeed. Mr. Morgan reduced it, with great ease, by gentle taxis. All the symptoms of strangulation of the intestine remaining unabated, Mr. Morgan examined the inguinal canal, and thought he felt a small tumour therein. He next cut through the integuments over the external ring and inguinal region, exposed the inguinal canal, and found a serous sac, which he opened, but he could not find the intestine. The patient died—about four days and a half from the commencement of the attack.

Necropsy.—The tumour which had been felt in the inguinal canal was a hernial sac, and it contained a loop of

[1] 'The Anatomy and Surgical Treatment of Abdominal Hernia,' folio, 2d edit., 1827, p. 83.

strangulated bowel. The impediment to the reduction of the hernia was the orifice of the sac. Immediately below this orifice was situated the opening of an abnormal pouch, which descended, downwards and inwards, behind the fascia transversalis, in the direction of the crural ring. It was into this pouch that a portion of the strangulated ileum had descended. The communication between the tunica vaginalis and the abdominal cavity was not completely closed, but the finger could not pass from the cavity of the tunica vaginalis into the peritoneal cavity because of the pressure exerted by the hernia.

Here is sufficient evidence that in this case the hernia had descended into the vaginal process of the peritoneum.

A preparation from this case is preserved in the museum of Guy's Hospital, and to make a careful examination it was removed from the bottle. The hernia, as before described, is situated between the fascia transversalis and peritoneum, and it has reached this situation through a rent in the neck of the hernial sac, which rent is the "opening of the abnormal pouch" already mentioned. The mouth of the hernial sac has been pushed away from the internal abdominal ring, and the laceration of the sac has taken place between these two points. The rent is on the posterior surface of the neck of the sac, and corresponds with the posterior and internal borders of the internal abdominal ring. I cannot discover a trace of those appearances which would result from "the hernial sac together with its contents" being pushed into the abdominal cavity. It maintains the same relations with the surrounding parts as it always had.

Case 5.—In June, 1851, a man was admitted under the care of Mr. Hilton, æt. 49. He was strong, healthy, and the subject of right, oblique inguino-scrotal rupture. He had been suffering with strangulated hernia some hours, when a surgeon succeeded, as he supposed, in effecting its reduction by the taxis. All the indications of strangulated

intestine existed on admission, although there was no tumour distinguishable in the scrotum, inguinal canal, or at the site of the internal abdominal ring. Mr. Hilton explored the inguinal region by a cutting operation, and, having opened the hernial sac in the inguinal canal, he discovered the strangulated bowel, divided the narrow mouth of the sac, and returned the intestine into the peritoneal cavity. The patient survived some hours, but succumbed under acute peritonitis.

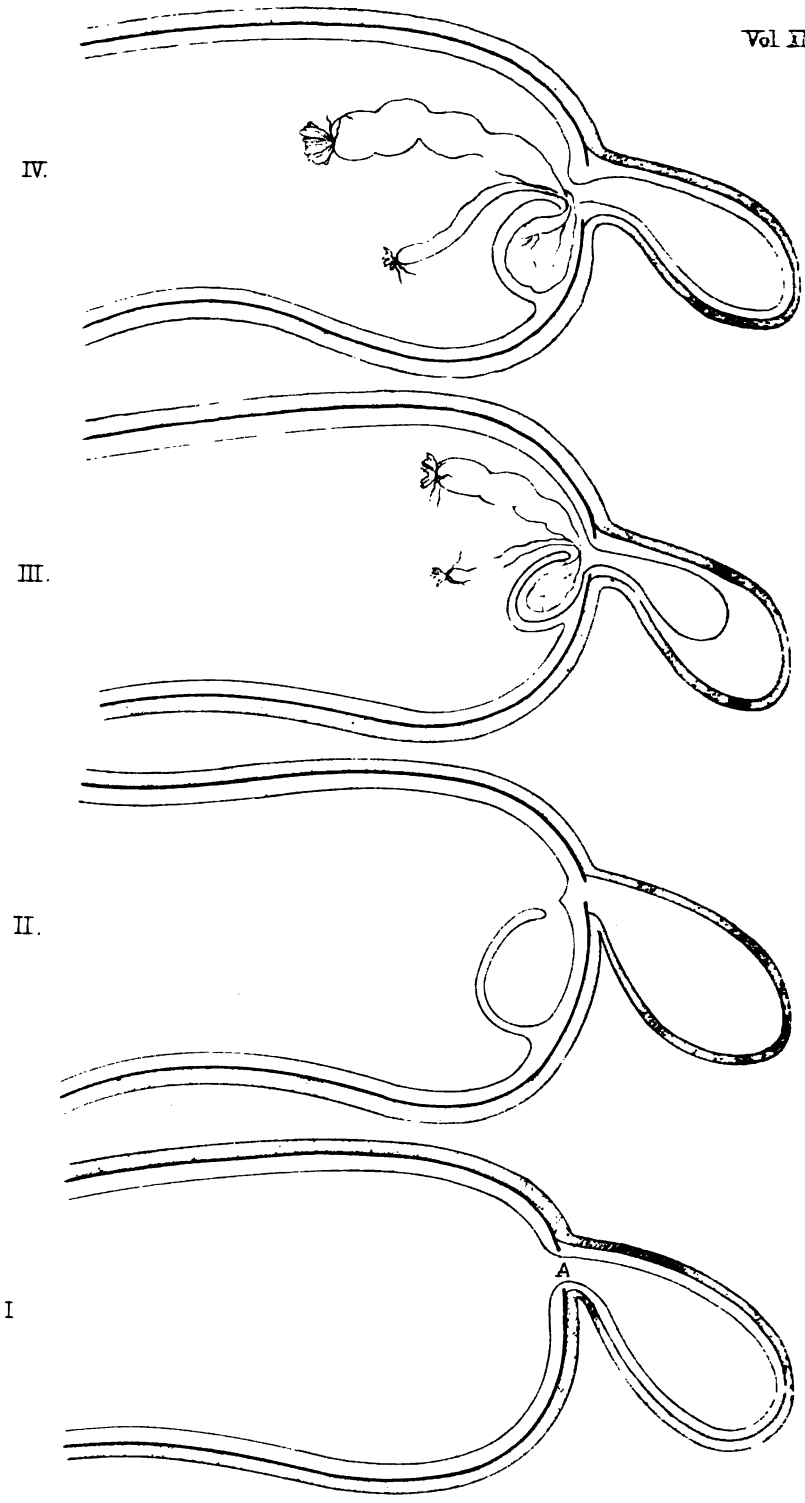
Necropsy.—Visible results of acute and general peritonitis. The hernia had been pushed out of the scrotum and inguinal canal through a lacerated opening in the neck of the hernial sac. It had been placed between the peritoneum and the internal abdominal fascia, the mouth of the hernial sac, whilst strangulating the bowel, having been likewise pushed away from its attachments to the internal surface of the internal abdominal ring. The body and fundus of the hernial sac still maintained their relations with the scrotum and inguinal canal.

Let me now explain my views of the mechanism of this accident.

Of the formation of the hernial sac.

In every observation or discussion concerning the varieties of oblique inguinal hernia, or of the accidents occurring in the treatment of such cases, it is imperative that a marked distinction be drawn between those cases in which the hernia descends into the vaginal process of the peritoneum, and those in which the sac is formed by a slow and gradual process of extension of the serous membrane from above downwards.

The shape, length, and relations of the several portions of the hernial sac are quite different in these two classes of cases, and the importance of always bearing in mind the marked differences between them will appear very prominently in some of the subsequent remarks I have to make.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However, I would here state that, in many of the cases in which a hernia descends into the vaginal process of the peritoneum, it only occupies at first the upper or inguinal portion of that sheath, and that contact with the testis and infantile development are not essential characteristics of this variety of oblique inguinal hernia.

Explanation of the Diagrams.

By the aid of diagrams I will now explain the mechanism of this accident, the so-called "réduction en bloc."

Diagram I—Describes a section of the abdominal cavity and scrotum; the broad, outside, crossed-surface, indicates the abdominal walls: within this the narrow, black line, represents the internal abdominal fascia, which constitutes the internal abdominal ring at A. The inner fine line indicates the relations of the peritoneal lining membrane passing into the scrotum through the internal abdominal ring.

This diagram represents the normal relations of a hernial sac to the abdominal walls and scrotum.

Diagram II.—This represents the relations of the peritoneal hernial sac, in accordance with the description which defines "réduction en bloc or en masse" to be "the reduction of the sac, together with its contents, within the abdominal walls."

The peritoneal sac only is represented.

The same parts are indicated as in the first diagram, and by similar lines.

Diagram III—Illustrates the morbid anatomy of the parts in the first case I have described. The knuckle of ileum is shown in the secondary sac or pouch, which has been formed by a dilatation of the neck of the hernial sac, after its orifice has been pushed away from the internal abdominal ring. A portion of the sac, which is empty, remains in the scrotum. The secondary sac, or pouch, is placed behind the internal abdominal fascia.

DIAGRAM IV.—In this diagram the orifice of the hernial sac is represented to be pushed away from the internal abdominal ring, the neck of the sac lacerated, and the hernia outside the peritoneum, between it and the internal abdominal fascia. In this variety of the accident the hernia is lodged in the loose subperitoneal connective tissue, and it has no serous sac investing it, as in the last variety.

The mechanism of the injury.

The mechanism of the injury, which seems then to be very simple, is as follows. By the pressure employed to reduce the hernia, the contracted orifice of the hernial sac, tightly embracing it, is pushed away from the internal abdominal ring; and, by the continuance of the pressure, the neck of the hernial sac becomes dilated into a pouch within the abdomen, which receives a part of the hernia. Now, if the pressure on the hernial tumour be continued, the neck of the sac becomes torn, and then the hernia escapes through the laceration, and is outside the peritoneum altogether.

The dilatation of the neck of the sac and its subsequent laceration are, I believe, very much favoured by the presence of fluid in the hernial sac, which then exerts an equal pressure on all sides, and that part which is the least strengthened by the surrounding structures is least able to resist it. With forcible pressure from the outside upon a sac containing fluid, if that fluid does not escape into the peritoneal cavity, through the mouth of the sac, the coats of the sac itself must at last give way, and, by this bursting of the investing sac, the fluid is allowed to extravasate into the neighbouring connective tissue. This gives rise to the delusive feeling that, as the hernial tumour is slowly diminishing in size, its contents must be passing into the peritoneal cavity; but such is not the fact.

Judging from the circumstance of the laceration being usually in the posterior wall of the neck of the sac, we may assume this part to be the weakest, and, examining it anatomically, it is certainly that part which derives the least strength from the surrounding structures.

At this point it becomes necessary to inquire how the secondary intra-abdominal pouch is formed. After a critical examination of all the recorded cases of reduction en masse, the fact is elicited that the accident has occurred in two distinct classes of cases, as follows, viz.:

1st. Those cases in which the reduction has been effected without much pressure; and—

2d. Those in which considerable force has been employed to push the hernia out of the scrotum.

In the cases of the first class, it is difficult to suppose that the gentle pressure employed could dilate the neck of the hernial sac into a pouch sufficiently large to contain the hernia, and therefore we must search in the history of these cases for an explanation of this phenomenon. The instances in which we may assume the gradual formation of such an intra-abdominal pouch are very few, and the patients had been the subjects of hernia for some years, and had worn trusses. I purposely employ the expression "assume," because there is no demonstration of the existence of such a pouch in any cases but those in which the "taxis" has been employed, and we well know that the pressure which one surgeon would call "gentle taxis," another would not describe by such a mild term.

Mr. Cock has offered an explanation of the formation of the intra-abdominal pouch in the paper to which reference has been already made.[1] After stating that there are two remaining forms to be described, he writes: "The first consists of the prolongation of the hernial sac beyond the internal ring into the abdominal cavity, where it becomes dilated into a pouch of greater or less size, lying on the fascia iliaca, between the internal ring and spinous process of the ilium, and contained between the fascia transversalis and the peritoneum. On the inner aspect of this pouch, at a distance of between one and two inches from the internal ring, is the opening of communication with the peritoneal cavity, and the circle of this opening forms the seat of stricture. This pouch is no doubt of slow and gradual

[1] 'Guy's Hospital Reports,' 1847, p. 54.

formation, and appears to be the result of frequent and protracted manipulation to reduce an old hernia, which has been in the habit of constantly descending through a lengthened period of time. The repeated application of the taxis, and the means constantly used by the patient to return the intestine, as often as it protruded, appear at length to have had the effect of separating the circle of peritoneum constituting the mouth of the sac from its connexion with the margin of the internal ring; a portion of the sac becomes pushed upwards from the inguinal canal through the opening in the fascia transversalis, and gradually dilates into the cavity, or pouch, which I have already described."

To this I will add another explanation. The pouch is situated between the mouth of the sac and the internal abdominal ring and inguinal canal. Now, the pad of the truss is commonly placed over the inguinal canal, and its pressure is successfully exerted to prevent the rupture from passing through the external abdominal ring. But, supposing the rupture to descend through the mouth of the sac, a double pressure will be exerted upon it, *below* by the pad of the truss, *above* by those forces which are continually pressing upon it from that region, and thus the constant influence of these forces would be upon a point of the hernial sac between the extremes, namely, its orifice and fundus. The point upon which the pressure would fall, will correspond to the neck of the hernial sac; it might then become dilated, and the intra-abdominal pouch would be developed.

However, the slow and gradual formation of such a pouch is purely conjectural, for its existence, independent of some complication which might be referred to the immediate application of the taxis, has not yet been recorded, to my knowledge.

The cases which might be quoted in support of the opinion that the intra-abdominal pouch is sometimes formed slowly and gradually, are as follows:

Arnaud, 'Diss,' p. 387, obs. ix, anno 1740.
Luke, 'Med.-Chir. Trans.,' xxvi, p. 162.
Cock, 'Guy's Hospital Reports,' 1847, p. 55.

But after a careful perusal of these cases it will be, I think, admitted that they scarcely uphold the theory.

PART III.

[In this Part an Analysis of the tabulated cases of Réduction en bloc is given.]

Ages at which the herniæ were developed.

Inguinal herniæ are developed in infancy, youth, and adult life.

Those occurring during the first and second periods usually descend into the vaginal process of the peritoneum, whilst those of the third period sometimes descend into the above-named peritoneal process, and are also formed as the result of a slow and gradual extension of the peritoneum.

Of the 37 cases, I am unable to state the age at which the hernia was developed in 15, but of the remainder it was formed at the respective ages as follows:

In infancy .	.	in 4 cases.
In youth .	.	in 2 „
After puberty—		
Before 20 years .	.	1 case.
From 20 to 30 years	.	8 cases.
„ 30 to 40 „	.	5 „
„ 40 to 50 „	.	2 „
		22
Not stated in	.	15
Total	.	37

From this table it should be observed how large a proportion of the cases of hernia, in which this accident (réduction en bloc) has resulted, was developed before thirty years of age, the proportion of the 22 cases being 15 before thirty years of age, and 7 after. This circumstance is significant of the precise nature of the hernia, which will be more fully developed immediately.

Of the variety of hernia.

All the cases were of the kind described as *oblique* inguinal, a very large majority being *scrotal*, that is, the hernia had passed through the external abdominal ring. A large majority was in the right side of the scrotum.

In the *right* side . .	20 cases.
In the *left* side . .	9 „
Not stated in which side .	8 „
	37

Some of the patients were afflicted with rupture on both sides, but the above table includes that side only, on which the hernia was reduced.

Numerical comparison between the cases of hernia into the vaginal process of the peritoneum and the variety of oblique inguinal hernia of slow development.—In a majority of cases the hernia descended into the vaginal process of the peritoneum, and of this fact the evidence is conclusive. The following table shows the numerical proportion:

Certainly into the vaginal process in	14 cases.
Probably into the vaginal process in	8 „
	22 „
Not sufficient evidence of being into vaginal process . . .	15 „
Total . . .	37 „

But even among the cases in the last category there are indications leading to the supposition, that even in a few of them the hernia may have been in this process of the peritoneum.

This circumstance is very important in regard to the mechanism of the accident. Bearing in view the anatomical differences, which exist between the cases in which the hernia descends along a canal already formed to permit its

descent into the scrotum, and those in which the hernia forms for itself a covering by pushing the peritoneum before it, we can readily understand why the laceration of the neck of the hernial sac takes place more frequently in the first class of cases than in the second.

Anatomical comparison between hernia into the vaginal process of the peritoneum and the variety of oblique inguinal hernia of slow development.—In cases of the *first* class, the orifice of the hernial sac is small, the situation in which it is originally formed is not influenced by the descent of the hernia, and the relations of the internal abdominal ring are maintained throughout, uninfluenced by the protrusion. Consequently, the length of that portion of the hernial sac lying in the inguinal canal, or between the internal and external abdominal rings, exceeds, very greatly, that of all other varieties of inguinal hernia. To this anatomical conformation the difficulty in reducing the hernia must be mainly attributed, for the orifice of the sac cannot either be felt or fixed, and any pressure from the scrotal region has a tendency to push the orifice of the sac away from the internal abdominal ring, especially too when the orifice so tightly constricts the hernia as to prevent it passing into the peritoneal cavity. This great length of the inguinal portion of the hernial sac would also favour its dilatation into a pouch within the internal abdominal ring, and the laceration of this, from prolonged pressure, would permit the escape of the hernia outside the peritoneum.

Now, the converse of all the anatomical relations pertaining to hernia into the vaginal process of the peritoneum is manifested in those varieties of inguinal hernia developed late in adult life, and preceded by a gradual extension of the peritoneal membrane to form the hernial sac. The orifice of the sac is large; the abdominal rings are approximated; the inguinal portion of the hernial sac is short; the orifice of the sac can be easily felt and fixed, and there is not the same facility offered to push the mouth of the

sac away from the internal abdominal ring, and lacerate its neck.

Situation of the testicle.

In three of the cases of hernia into the vaginal process of the peritoneum, the testis of the same side as that in which the hernia was formed, was lodged in the inguinal canal. In two of these, the organ had never descended into the scrotum, but in one it is stated that, upon an occasion when the rupture had been reduced by much force, the testicle, together with the hernia, had been pushed back into the abdomen.

By whom, and by what local means the hernia was pushed out of the scrotum, and the nature of the constitutional measures employed to assist in the reduction.

In some of the cases a surgeon, and in others the sufferer, effected the removal of the hernia from the scrotum. The force employed is variously described, thus: "by moderate pressure," "with great difficulty," "avec beaucoup de peine."

The constitutional means have been those usually adopted in cases of strangulated hernia—hot baths, venesection, opium, and chloroform. I believe that it is during the influence of this anæsthetic agent that great mischief may be done, and that the sac may be very easily torn at the time when all muscular tonicity is removed.

Local indications that the hernia is not reduced into the peritoneal cavity.

In the majority of the cases there has been usually some local indication that the hernia was not entirely reduced into the peritoneal cavity. These were repeated descents of the protrusion, slight fulness at the internal ring, and pain on pressure over this region. Even a small

persistent tumour has, in a few cases, indicated the necessity for exploration.

PART IV.

Practical deductions.

Very important practical deductions are involved in the preceding analysis of the cases, and I shall now endeavour to extract those facts which must exert important influences in the formation of a correct diagnostication of these complicated cases, as well as on their successful treatment.

I. *Diagnosis.*

1. The analysis shows that cases of this nature may occur in patients varying between the ages of thirteen years and seventy-nine. Therefore, at any age after puberty, and during adult life, the surgeon may be prepared to meet with this accident.

2. Complications arising from the lesion described seem to affect the herniæ developed in early life more frequently than those which are formed after forty years of age. And thus the diagnostication may be assisted by ascertaining what was the age of the individual under observation at the time when the rupture was first developed.

3. All the observations on record were of *oblique* inguinal herniæ, a majority of them being scrotal, and on the right side. When there is a rupture on both sides of the scrotum, the diagnostication of the strangulated bowel is rendered extremely difficult. But, acting upon the experience of the successful issue of a case in which Dupuytren opened both the inguinal regions, the surgeon would be justified in following the example.[1] In one case,

[1] Dupuytren, 'Leç. Orales,' edit. 1832, t. i, p. 583.

a correct diagnostication was formed of the side upon which the strangulated bowel existed, by observing that on one side the rupture readily descended, whilst on the other it seemed to be reduced, and could not be made to come down, which it had always previously done upon exertion. This was the side upon which the bowel was strangulated, and an operation saved the life of the patient.[1]

4. A large proportion of the cases of oblique inguinal hernia, to which this accident happened, was of that class in which the bowel descends into the vaginal process of the peritoneum. Accuracy in diagnostication might, therefore, be assisted by obtaining a correct history of the mode of development of the rupture; whether it appeared suddenly in the inguinal canal or scrotum, as the result of immediate exertion or violence, or was the production of slow and long-continued pressure from above, and relaxation of the tissues of the region.

5. The situation of the testis may be a cause of obscurity in the diagnostication of these cases of hernia. When congenitally absent from the scrotum, it is often in the neighbourhood of the abdominal rings, and forms a swelling, which, from the pain caused by pressure over it, might mislead the surgeon as to its precise nature. A careful examination of the region, when a deformity of this kind exists, can alone place the nature of the case clearly before the observer.

6. In the majority of these cases the hernia consisted of reducible intestine *only*, and therefore, when this was pushed outside the sac, and behind the peritoneum, nothing but the sac itself remained as an additional element to the tissues of the scrotum. Under these circumstances, until it can be proved that the hernial sac had been torn away from its scrotal attachments, it must maintain its relations with them. The assumption of the translation of the hernial sac from the scrotum into the abdomen is based upon very insufficient evidence, and, until ocular demonstra-

[1] Dupuytren, 'Leç. Orales,' edit. 1832, t. i, p. 587.

tion of the fact exists, we may expect to find, certainly in scrotal herniæ, some trace of the hernial sac at the upper part of the scrotum, or in the inguinal canal. A majority of the cases show this to be an indisputable fact. But in those cases of entero-epiplocele, in which the omentum is adherent to the hernial sac, it remains irreducible after the reduction of the bowel, and then more or less swelling is perceptible in the inguinal region. I dwell particularly upon these facts, because Mr. Luke, in his very practical paper, writes that "the absence of such fulness in a part, when hernia is known to have previously descended, necessarily leads to the conclusion, that the sac upon which it depended has been displaced, and probably returned, together with the hernia."[1] If, therefore, too much reliance be placed upon the absence of all local indications of a hernia having existed, an incorrect diagnosis might be formed of those instances in which adherent and irreducible omentum is present.

7. A disposition to the recurrence of the hernia may exist many years before the accident which is the subject of this paper happens to it. Or, in other words, herniæ of quite recent formation, as well as those of long standing, may be reduced "en bloc;" that is, the hernial sac may be ruptured in the application of the taxis, and its contents escape through the laceration.

8. The production of this injury must be the result of the application of more or less violence, in the majority of cases, if my interpretation of the nature of the local lesion be correct. When all the tissues of the part are relaxed, and all muscular tonicity and resistance overcome by the anæsthetic influence of chloroform, or any means giving rise to conditions closely approximating such results, it needs but very little pressure to be exerted in order to tear the peritoneal sac, especially when circumstances, before alluded to, favour such lesion. In the diagnostication, then, of this injury to the hernial sac, if too much reliance be placed

[1] 'Medico-Chirurg. Trans.,' vol. xxvi, p. 175.

upon the statements of the employment by the operator of "moderate pressure," "gentle taxis," "no force," and expressions of like tendency, the surgeon may be led astray.

9. Are there any local indications by which the surgeon may diagnosticate the nature of this lesion, and the situation of the hernia? In the majority of the recorded cases the writers describe local indications more or less marked.

They are as follows:

a. Repeated descents of the hernia after its reduction is supposed to have been accomplished.

b. Swelling in the iliac fossa.

c. A hard tumour *to be felt* in the inguinal region and canal.

d. Slight protrusion at the external ring after making efforts to produce the rupture.

e. Pain on pressure and fulness in the iliac fossa.

f. A tumour to be indistinctly felt at the internal abdominal ring.

10. In all the cases the constitutional indications of strangulated intestine were persistent.

II. *Treatment.*

A review of the cases on the tables very clearly establishes the rule to be followed in the treatment of these cases. If the strangulated bowel is not relieved from its constriction, and the impediment to its reduction into the peritoneal cavity removed, the death of the sufferer is imminent, nay, certain!

To avoid, in the first place, the risk of the occurrence of the complication, great care and circumspection are required on the part of the employer of the taxis, especially *if the patient be under the influence of chloroform*, and the case in hand should be of that variety of hernia which descends into the vaginal process of the peritoneum. This last fact must be ascertained by careful and minute inquiry into the *early* history of the case, the manner of the production of the

hernia, and the age of the patient at the time of its development.

Should there be even the slightest indications of the complication described in this paper, the operator must proceed to explore the inguinal region, for delay is inadmissible. In this operation he should first search for the hernial sac. Having opened this, upon passing the finger upwards to the internal ring, he will very probably pass it through the laceration in the sac, and, feeling intestine, he will suppose that he has reached the peritoneal cavity. This, however, he has not yet accomplished. He must next draw the bowel into the inguinal canal, and then, by passing the finger along the *anterior* surface of the mesentery, he will reach *the orifice* of the hernial sac, which firmly constricts the protrusion. The orifice of the sac must now be divided, and in the reduction of the hernia into the peritoneal cavity, great care is required to prevent it gliding through the laceration. The division of the constriction, or orifice, of the hernial sac is accomplished with greater facility, if an assistant steadily draws the sac downwards, whilst the operator introduces the bistoury on his index finger.

This communication having extended far beyond the limits which I contemplated at its commencement, an apology is due to the Society, and, in relying upon the forgiveness of its members, I trust they will not accuse me of endeavouring to support a theory, for my sole design has been the discovery of the truth, and therefore the advancement of practical surgery.

TA

Cases of Inguinal Hernia re

Case.	Sex.	Age of Patient.	Variety of Hernia. Usual size.	Right or Left.	Age of Hernia.	Age at development.	By whom reduced.	Means employ
1	M.	50	Enterocele	...	"Years"	—	A surgeon	—
2	M.	13	Enterocele. Probably in vaginal process	—	—	—	—	—
3	M.	47	Enterocele; scrotal. Probably in vaginal process	Right	20 years	27	A surgeon	—
4	M.	67	Enterocele; scrotal	Right	30 years	37	Surgeon	Hot bath which indu faintness
5	M.	30	Enterocele; scrotal. Probably in vaginal process	Right	5 years	—	Surgeon	—
6	M	—	Enterocele; congenital. In vaginal process	—	—	—	—	—
7	M.	42	Enterocele; scrotal; size of hen's egg	—	"Years"	—	Surgeon	After venesec tion
8	M.	—	Enterocele; scrotal; size of hen's egg	—	"Years"	—	Patient	—

oc." No operation. Death.

l indications of after reduction.	Survived this attack.	Necropsy.	Anno.	Reference.
None	Reduction; six months. Last severe attack eight days	The intestine was not under strangulation from the ring. A bag about the bulk of a hand-ball, formed by peritoneum, contained three inches of mortified bowel (ileum)	1740	Arnaud; Diss., p. 387, obs. ix.
None	—	"The intestine, still strangulated by the neck of the hernial sac, had been pushed up along with the sac beyond the ring, where it was seen rolled up between the aponeurotic parietes of abdomen and peritoneum"	1818—1819	Scarpa; Sull'ernie, 2d edit., p. 49.
eated descents	About 3 days	Hernia in scrotum; it had been pushed within the abdominal muscles, but not within the peritoneum; neck of sac torn from internal ring	1828	Sir C. Bell; Lond. Med. Gaz., 1828, vol. i, p. 484.
None	—	A knuckle of intestine was discovered in a sac, lying between right linea ilio-pectinea and bladder	1834	B. B. Cooper; Guy's Hosp. Reports, iv, p. 331.
—	6 to 7 days	Knuckle of jejunum confined in inner portion of abdominal ring, strictured by hernial sac	1836	Parish; Observations on Strangulated Hernia.
—	—	"*Arrachement du collet*" of the hernial sac, by which the bowel was strangulated within the abdomen	1840	Laugier; Bull. Chir., i, p. 363.
None	About 4 days	A small knuckle of intestine just inside the inner ring, strangulated by the sac, which had been returned with it.	1842	Banner; Prov. Med. and Surg. Jour., 1843.
None	About 4 days	—	1844-5	Blackman, p. 38.

TABL

Cases of Inguinal Hernia reduc

Case.	Sex.	Age of Patient.	Variety of Hernia. Usual size.	Right or Left.	Age of Hernia.	Age at develop. ment.	By whom reduced.	Means employed
1	M.	38—40	Enterocele; scrotal. Seven or eight inches long. Probably in vaginal process	Right	15 years	23—25	Surgeon	—
2	M.	40	Entero-epiplocele; double hernia. Probably in vaginal process	Left	12 years	28	Surgeon	"Beaucoup de peine"
3	M.	—	Enterocele, in contact with testis. In vaginal process	—	—	Infancy	Surgeon	—
4	M.	23	Enterocele; *not* in scrotum; *first descent*. In vaginal process	Right	A few hours	23	Surgeon	—
5	M.	—	Enterocele; double hernia	Right	11 years	—	Patient	—
6	M.	39	Enterocele; testis in inguinal canal. In vaginal process	Right	"Years"	—	Surgeon	"Moderate pressure"

[1] Arnaud makes this observation (p. 375): "I tried again to bring out the rupture, b contiguous parts upon which it lay, confined it so much that it could not be moved."

en bloc." *Operation.* *Recovery.*

Local indications of Hernia after reduction.	Duration of strangulation.	Nature of Operation.	Anno.	Reference.
None perceptible to sight. Could feel a hard tumour in inguinal region and canal	Between 3 and 4 days	External ring incised; hernial sac opened in inguinal canal; orifice of sac very deep, but incised, and bowel relieved.[1] The tumour ran along the rectus	1736	Arnaud; p. 371, obs. v.
None in *left* inguinal region. Tumour in *right*	Between 5 and 6 days	*Right* sac opened first, empty. *Left* next, sac opened in inguinal canal; contents, bloody serum, omentum, and bowel; orifice cut, bowel reduced	1814	Dupuytren; Lec. Orales, ed. 1832, t. i, p. 583.
A tumour could be felt in inguinal canal	—	Sac opened in inguinal canal[2]	18—?	Sabatier; edit. 1824, t. iii, p. 520.
Swelling in iliac fossa	—	Inguinal canal exposed, and tumour appeared at internal ring; the hernial sac was opened, orifice cut, and bowel reduced	1824	Sabatier; edit. 1824, iii, p. 522.
Only "une espèce de cordon cylindrique"	About 2 days	Sac opened in inguinal canal, and a *cul-de-sac* felt higher up; orifice of sac cut, bowel reduced	1832	Dupuytren, p. 587.
"Slight fulness" in right inguinal region; small flaccid tumour at external ring when erect; tumour in site of internal ring which was testis	About 24 hours	Sac opened in inguinal canal, testis seen, and sac with contents drawn out from abdomen; orifice of sac incised, and bowel returned	1843	Luke; Med. Gaz., Nov., 1843.

with as little success as before; the adhesions which the sac had contracted with the

[2] I have doubts of this being a case in point.

Case.	Sex.	Age of Patient.	Variety of Hernia. Usual size.	Right or Left.	Age of Hernia.	Age at development.	By whom reduced.	Means employed.
7	M.	40	Enterocele; scrotal; double. Ruptured on right side before 20. In vaginal process	Left	4 years	36	Patient	—
8	M.	74	Enterocele, size of a good sized pear; double	Right	30 years	44	—	—
9	M.	77	Enterocele	Left	"Years"	—	Patient	—
10	M.	50	Enterocele; "in iliac region"	Right	11 years	39	Surgeon	—
11	M.	62	Entero-epiplocele; not in scrotum. In vaginal process	Right	62 years	From infancy.	Patient and surgeon	—
12	M.	—	Entero-epiplocele; scrotal	—	—	—	Patient	—
13	M.	14	Enterocele; in vaginal process	Right	2 or 3 days	14	Surgeon	Chloroform

Local indications of Hernia after reduction.	Duration of strangulation.	Nature of Operation.	Anno.	Reference.
Not a trace at operation; there had been a little fulness before	44 to 45 hours	Inguinal canal laid open, sac found at internal ring, cut, stricture divided, and bowel reduced	1843	Luke; Med.-Chir. Transact., xxvi, p. 166.
Slight protrusion at external ring after efforts to produce the rupture	About 3 days	Sac opened in inguinal canal, and bowel exposed; orifice of sac cut, and bowel reduced	1845	Wade; Lancet, 1845, July.
None, except slight pain on pressure above left inguinal canal	Nearly 6 days	Inguinal canal opened, and cord exposed. Tumour felt at internal ring, opened; hernial sac traced to its orifice which was cut, and bowel reduced	1849	Luke; Med. Gaz., 1850, p. 236.
None	14 hours	Inguinal canal opened, and hernial sac found at internal ring, pulled down, cut, orifice of sac dilated, and bowel reduced	1849	Syme; Monthly Jour. of Med. Science, x, p. 1, Jan., 1850.
Pain in iliac fossa, which was fuller than *left* side	About 3 days	Inguinal canal laid open; hernial sac felt in abdomen, exposed, incised at fundus and orifice, bowel reduced	1849	Melchiori; Gazette Med. Lomb., Nov. 1849.
None	—	Inguinal canal laid open; hernial sac in abdomen, incised in body and orifice, bowel reduced	1849	Robert; L'Union Med., 1849.
Tumour indistinctly felt at internal ring	2 to 3 days	Inguinal canal opened; sac pulled down, its orifice and ring divided, bowel reduced	1854	Ward; Memoir on Strangulated Hernia, p. 20.

TABL

Cases of Inguinal Hernia reduc

Case.	Sex.	Age of Patient.	Variety of Hernia. Usual size.	Right or Left.	Age of Hernia.	Age at development.	By whom reduced.	Means employed.
1	M.	—	Enterocele	—	—	—	—	—
2	M.	40	Entero-epiplocele; scrotal. In vaginal process	Right	30 to 40 years	In youth	No account	—
3	M.	55	Enterocele; congenital. Double hernia, scrotal, size of pigeon's egg. In vaginal process	Left	55 years	Infancy	Patient	—
4	M.	58	Enterocele; scrotal	Right	"Years"	—	Surgeon	—
5	M.	30	Enterocele; scrotal; "large." Probably in vaginal process	Left	"Years"	—	The patient, with very little force and not the slightest pain	—
6	M.	34	Enterocele; scrotal. In vaginal process. Testis in inguinal canal	Right	A few hours	34	Surgeon	—
7	M.	64	Entero-epiplocele; scrotal. Probably in vaginal process	Left	36 years	28	Surgeon	Warm bath, opium, ice

II.

en bloc." *Operation.* *Death.* *Necropsy.*

Local indications of Hernia after reduction.	Duration of strangulation.	Nature of Operation.	Necropsy.	Anno.	Reference.
—	—	Sac opened in inguinal canal; orifice not incised	Hernia strangulated by orifice of sac, which was on psoas magnus	1740	Vacher, Arnaud; Diss., p. 386, obs. viii.
A tumour	—	Sac opened in scrotum and inguinal canal; omentum in sac; no intestine seen	A sort of double sac, one external to the other, inside the abdomen, which contained ileum	1801	Hernu; Recueil périodique de la Soc. de Med., 7 Messidor, an IX (1801), t. xi, p. 291.
Pain and fulness in left inguinal region	5 days	Inguinal canal opened; sac opened in canal; the true sac in abdomen; incised; orifice cut; bowel reduced	No account of the parts	1817	Dupuytren; Jobert; Mal. Chir. du canal intestin, i, 492.
Small tumour in canal	4 days	Sac opened in canal; no bowel	A pouch extended downwards and inwards behind fasc. trans. in direction of crural ring	1825	Astley Cooper on Hernia, 2d edit., p. 83.
Tumefaction in canal after eight days. None before	8 days	Sac opened in canal; orifice not incised	Large hernial sac just within abdominal walls, its orifice away from internal ring	1839	Luke; Med.-Chir., xxvi, p. 162.
Tendency to reproduction of tumour after taxis	8 days	Sac opened in scrotum and inguinal canal; no bowel; sero-albuminous fluid	Portion of ileum in iliac fossa, probably outside peritoneum	1839	Parise; Bérard; Mém. de la Soc. de Chir. de Paris, t. ii, p. 399.
Tumour from omentum	8 days	Sac opened in canal	Pouch in abdomen, constricting bowel	1847	Guy's Hos. Reports, 1847, p. 56.

Case.	Sex.	Age of Patient.	Variety of Hernia. Usual size.	Right or Left.	Age of Hernia.	Age at development.	By whom reduced.	Means employed.
8	M.	35	Entero-epiplocele; congenital. In vaginal process. Testis in inguinal canal	Left	35 years	Infancy	Surgeon	—
9	M.	30	Enterocele. In vaginal process	Right	10 or 12 years	18—20	Patient	—
10	M.	79	Entero-epiplocele; scrotal	Right	30 years	49	Surgeon	—
11	M.	75	Enterocele	Right	40 years	35	—	—
12	M.	35	Enterocele. Probably in vaginal process	Right	"Years"	—	Surgeon	—
13	M.	49	Enterocele	Right	—	—	Surgeon	—
14	M.	14	Entero-epiplocele; congenital; scrotal. In vaginal process	Right	2 years	Childhood	Surgeon	—
15	M.	37	Enterocele; scrotal; congenital; double. In vaginal process	Left	9 or 10 years	27	Surgeon	"With great difficulty"
16	M.	51	Enterocele; scrotal; double	Left	—	—	Patient	—

Notes and observations.—Case 1. The hernia seems

Local indications of Hernia after reduction.	Duration of strangulation.	Nature of Operation.	Necropsy.	Anno.	Reference.
Only an empty sac	—	Sac opened in canal	Pouch in abdomen, &c.	1847	Idem, p. 55.
Descent of hernia	4 or 5 days	Sac opened in canal; orifice of sac cut; hernia reduced	Laceration of neck of sac; hernia between peritoneum and internal abdominal fascia	1847	Idem, p. 66.
Small and soft tumour at ring	25 hours	Sac opened, and orifice cut	None	1848	Luke; Med. Gaz., 1849, p. 272.
—	—	—	Laceration of sac; hernia between peritoneum and internal abdominal fascia	1849	Reid; Prov. Med. and Surg. Journal, 1849.
No history of a hernia having descended	6 days	Inguinal canal opened; hernial sac at internal ring; incised; bowel escaped; stricture divided	Acute peritonitis; hernial sac and contents between peritoneum and fasc. trans.	1850	Curling; Lancet, 1850, ii, p. 81.
Fulness in inguinal canal	3 to 4 days	Sac opened in inguinal canal; hernia seen; stricture cut; hernia reduced	Acute peritonitis; neck of hernial sac torn, and bowel outside peritoneum between it and internal abdominal fascia	1851	Personal obs.
Fulness in inguinal canal	3 to 4 days	Hernial sac opened in inguinal canal; bowel thought to be reduced through its orifice	Neck of hernial sac torn; bowel lodged between peritoneum and internal abdominal fascia	1854	Personal obs.
—	—	Hernial sac opened in inguinal canal; stricture at orifice	Orifice of sac divided by operator a little above internal ring	1854	Ward; 1st Mem., p. 22.
Obscure swelling at upper part of inguinal canal	7 or 8 days	Hernial sac opened in inguinal canal; orifice cut	"Neck of sac" over border of inner and anterior part of true pelvis; body of sac in iliac fossa	1854	Ward; 1st Mem., p. 21.

to have been reduced through the *external* ring only.

ON

THE ADMINISTRATION OF BELLADONNA,

AND ON

CERTAIN CAUSES WHICH MODIFY ITS ACTION.

BY

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IN the month of November, 1858, my attention was arrested by the extraordinary tolerance of belladonna exhibited by a child to whom I was administering it as a remedy for chorea. Commencing with a quarter of a grain of the extract administered in divided doses daily, I gradually increased the quantity of the drug up to six grains a day, with no other effect than that of arresting the choreic spasm and producing very slight dilatation of the pupil. I therefore determined to institute some experiments, with the view of ascertaining whether the case in question was exceptional, or whether chorea does not exercise some modifying power in respect to the action of the drug. The results, which to me are novel and remarkable, form the subject of the present communication.

The cases on which my observations were made are twelve in number. The patients were female children varying in age from eight to nineteen; three of them had

previously experienced one or more attacks of chorea, and in five of them the symptoms of the present attack were of unusual severity. In every instance the involuntary movements were general, affecting the extremities on both sides of the body as well as the muscles of the face; in four instances the patients were unable to stand, and in three instances the violence of the jactitations rendered it necessary for the patients to be tied in bed. In eleven out of the twelve cases the disease came on spontaneously, without fright or previous illness; in the other (Case 9) the symptoms almost immediately followed the shock produced by seeing her father brought home a corpse.

In the first eleven cases belladonna was given, and the extract was the preparation employed. In the first case the drug was given in pills; in the second case half the quantity was administered in pills and half dissolved in water; in the remaining nine cases the whole quantity prescribed each day was dissolved in two ounces of water, of which half an ounce was given as a dose every six hours.

In nine of these cases the hospital extract of belladonna was used. This is obtained from Apothecaries' Hall. Its power was tested as follows: a solution of one grain to a drachm of water dropped into the eye of my clinical clerk caused enormous dilatation of the pupil, which lasted two days; a grain and a half given twice a day to a male adult suffering from sciatica produced so much vertigo and dryness of the throat and fauces, that the man was unable to continue taking it; a grain and a half taken at a dose experimentally by our Medical Registrar gave rise to dryness of the throat and fauces, and dilatation of the pupil. However, with the view of obviating any objections as to the strength of the drug, the extract obtained from J. Bell's, in Oxford Street, was administered in Case 5, and that procured from Squire's, in Oxford Street, in Case 7.

In Case 12 atropine was employed. The alkaloid was obtained at Morson's, in Southampton Row, and was dissolved by means of a few minims of the dilute sulphuric acid

in two ounces of water, of which half an ounce was given four times daily.

In neither of the twelve cases was any other remedy given except an occasional dose of castor oil, or senna draught, or a few grains of scammony and calomel when the bowels were costive.

The diet, except in those cases in which the appetite was indifferent, or in which the choreic spasm was so violent as to prevent mastication, consisted of bread, meat, and vegetable—the hospital "ordinary diet;" in the others, of beef-tea, eggs, and the hospital "milk diet."

The following brief outline of the cases will suffice for the purpose of this communication. They are given in chronological order.

Case 1.—Alice S—, æt. 11, was admitted into the Crayle Ward of St. George's Hospital, November 17th, 1858; suffering from chorea, affecting the muscles of the face and of both upper and lower extremities. She had experienced a severe attack of the disease, which nearly proved fatal, in the month of June previously, but had been relieved by sulphate of zinc administered in rapidly increasing doses. Her present symptoms commenced at the end of October, and had gradually increased up to the date of her admission. There was no cardiac murmur. She was very pale. The pupils were large. As the sulphate of zinc had proved so useful on the former occasion it was ordered again, in combination with quinine and sulphate of iron, and its dose was rapidly increased, so that by the 25th of November eight grains were being taken three times a day. At the same time the bowels were freely acted on by means of scammony, calomel, and castor oil. Nevertheless little or no improvement took place in the choreic symptoms, so on the 27th the medicine was omitted, and a quarter of a grain of the extract of belladonna was given daily. After a time this dose was given twice, then thrice, and then four times daily, and it was cautiously increased

until, on the 14th of December, six grains of the extract were taken in the course of the twenty-four hours. On the 21st the notes of Mr. Seaton, my clinical clerk, report the patient as "quite steady," and on the 28th she left the hospital. Slight dilatation of the pupil was the only obtainable evidence of the action of the belladonna.

Case 2.—Ann B—, æt. 16, was admitted into Queen's Ward, November 17th, 1858, suffering from chorea of two weeks' duration. The spasms affected all the extremities; the face also was affected, and she was unable to articulate. The pupils were large; there was no cardiac murmur. The infusion of valerian with sulphate of zinc in solution was given every six hours, and the dose of zinc was rapidly increased, so that by the 2d of December fifteen grains were being taken four times daily. By the 4th, however, very little benefit had resulted, so on that day the medicine was omitted, and half a grain of extract of belladonna was given night and morning. On the 7th two thirds of a grain were prescribed three times a day, and on the 11th a grain was given four times daily. From this time the dose was rapidly increased, so that by the 16th of January fourteen grains were taken daily. By that time the spasms had almost ceased, and the patient could speak fairly. On the 17th she complained of nausea and the urine was ammoniacal. On the 18th she vomited, the bowels were much purged, and the pupils dilated. The spasms had entirely ceased. The medicine was omitted, but no other remedy was resorted to. She remained in the hospital another fortnight, in order that we might ascertain whether there would be any return of spasm, and as that was not the case, she was discharged on the 2d of February.

Case 3.—Jane C—, æt. 12, was admitted into Hollond Ward, January 12th, 1859. She had been suffering two months from chorea affecting the face and all the extremities. She had never experienced an attack of rheumatism,

but a loud systolic murmur was audible at the base of the heart, whilst at the apex an intensely loud murmur accompanied the systole, and sometimes also the diastole of the heart. Fremitus could be felt over the lower half of the præcordial region. The pupils were large. A quarter of a grain of the extract of belladonna was given four times daily, and the dose was gradually increased until, on the 7th of February, eleven grains were taken in the course of the twenty-four hours. The choreic spasms had almost wholly ceased. On the 8th the patient was sick in the morning, the bowels were relaxed, and she complained of lassitude and headache. The pupils, however, were not much larger than natural, there was not any indistinctness of vision, and the tongue was clean and moist. The cardiac murmurs were still audible, though not so loud as on admission; the heart's action was quieter, and the fremitus had ceased. No more medicine of any kind was given, and she left the hospital, on the 15th of February, perfectly free from chorea.

Case 4.—Harriet H—, æt. 19, was admitted into Crayle Ward on the 26th of January, 1859. The muscles of the face and all the extremities were affected with choreic spasm, and the fingers of the right hand were spasmodically closed. The pupils were large, the heart's sounds clear, the catamenia regular. Two grains of the extract of belladonna were ordered, and the dose was gradually increased until, by the 4th of February, she was taking seven grains daily. On the 5th I find a note by Mr. Seaton, "She is quite steady," and on the 8th she left the hospital.

Case 5.—Mary Ann F—, æt. 12, was admitted into Rosebery Ward on January 26th, 1859. Four years previously she had been in St. George's Hospital with a severe attack of chorea, which nearly proved fatal. The present attack commenced six weeks prior to her admission, and the symptoms had gradually increased in severity. The

muscles of the face and of all the extremities were affected, but those on the left side most severely. Two grains of the extract of belladonna were administered at first, and the dose was gradually increased until, by the 2d of March, she was taking forty-eight grains daily. She was then attacked with vomiting and purging, and thereupon the medicine was omitted. No other remedies were employed, but the symptoms of chorea subsided rapidly; indeed they almost ceased from the date of the vomiting and purging, and she left the hospital on the 9th of March in good health.

Case 6.—Jane S—, æt. 9, was admitted into Pepys' Ward on February 1st, 1859. She had been suffering from chorea about two months, and the symptoms had increased during the last three weeks. All the extremities were affected with spasm, and the muscles of the face slightly so. The heart's sounds were clear, the pupils large. Two grains of the extract of belladonna were ordered, and the dose was gradually but rapidly increased, so that by the 12th ten grains were taken daily. By this time the choreic movements had almost ceased, and the patient left the hospital perfectly well on the 16th.

Case 7.—Ann W—, æt. 11, was admitted into Crayle Ward on February 12th, 1859. The choreic symptoms, which were very severe, commenced about one month previously, and had gradually increased in severity. The whole of the limbs were in a state of violent jactitation, the skin of the hands and arms was rough, from the continued friction, and it was found necessary to tie her in bed. She was unable to speak, and could with difficulty swallow, or protrude the tongue. The pupils were large, and as far as could be ascertained, the heart's sounds were free from murmur. Four grains of the extract of belladonna were given, and the dose was rapidly increased until, by the 12th of March, sixty-eight grains of the extract were taken daily. By this date the choreic spasms were comparatively

slight, the patient could protrude the tongue and swallow without difficulty, and she was beginning to speak. On the 13th her bowels became relaxed, the motions being dark coloured, thin, and offensive; she complained of nausea, and was sick after her meals. On the 14th these symptoms had increased, and therefore, although the pupils were of their natural size, the sight was unaffected, and the tongue clear and moist, it was deemed expedient to omit the medicine. She now took no medicine for a fortnight, but the spasms continued to decrease, and the power of speech began to return. She was still unable to stand, in consequence of the choreic movements of the lower extremities. On the 28th, as she was pale and weak, a grain of sulphate of iron, and the same quantity of sulphate of zinc, was given every six hours, and the quantity of sulphate of zinc was rapidly increased, so that by the 16th of April she was taking twenty grains at a dose. Day by day the spasms subsided and her strength returned, and she left the hospital on the 20th of April in good health, and free from choreic symptoms.

Case 8.—Catherine L—, æt. 10, was admitted into Hollond Ward on February 16th, 1859. She had experienced two attacks of rheumatic fever, from the last of which she recovered five weeks before her admission into the hospital. On its cessation, symptoms of chorea began to manifest themselves, and gradually increased up to the date of her admission. The pupils were of medium size. A slight systolic murmur was audible at the apex of the heart. Four grains of extract of belladonna were prescribed, and day by day the dose was increased until, by the 2d of March, thirty-four grains were being taken daily. By this time the choreic symptoms had disappeared, and it was agreed that in a few days the patient should leave the hospital. On the evening of the 3d, however, she was attacked with diphtheria; the belladonna was omitted, and quinine with muriated tincture of iron and wine were substituted for it. On the 19th the syrup of iodide of

iron and iodide of potassium were given, with the view of getting rid of some enlargement of the cervical glands which remained after the throat affection had been subdued, and she was made out-patient on the 23d of March. There was not any recurrence of the choreic symptoms. The systolic murmur was still audible, though not so loud as on her admission.

Case 9.—Grace S—, æt. 10, was admitted into Pepys' Ward on February 18th, 1859. She had been ill a fortnight with choreic symptoms of the most violent description, which had followed immediately the shock produced by seeing her father brought home a corpse. The limbs were in a state of incessant jactitation; there was continued and violent grinding of the teeth; she had been unable to sleep or to utter a syllable since the beginning of the attack, and she swallowed with the greatest difficulty. The pupils were of medium size, and, as far as could be ascertained, the heart's sounds were clear and unaccompanied by murmur. The skin of the face, arms, and trunk, was excessively rough from continual friction, and the lips were chapped. The pupils were of medium size. Six grains of the extract of belladonna were ordered, and the dose was gradually increased, so that by the 23d twelve grains were taken daily. At this time the spasms appeared to be somewhat less severe, and the patient for the first time obtained a little quiet sleep. But the lull was of very short duration; as soon as she awoke the spasms recurred with increased violence. By the 2d of March the dose of belladonna had been increased to thirty-three grains daily, but the spasms were almost as severe as ever, the grinding of the teeth was incessant, there was the greatest difficulty in getting even liquid nourishment between her teeth, and she got no sleep. Moreover, the whole body was rough and sore from continued friction; the lips were deeply fissured; the face, chin, and upper part of the trunk, were covered with pustules, the result of friction; an abscess was forming on the neck; the skin over the right hip was abraded; and

the skin of the back was beginning to slough. On the 3d these symptoms had increased, and, in spite of wine which was administered freely, she sank exhausted on the following day. She was perfectly conscious throughout, and even two hours before her death recognised her friends. Unfortunately they would not permit a post-mortem examination.

Case 10.—Joanna G—, æt. 11, was admitted into Crayle Ward on February 23d, 1859. She had been suffering from chorea more or less for three months, and had been much worse during the last ten days. Her pupils were large; the heart's sounds free from murmur. Four grains of the extract of belladonna were prescribed, and the dose was gradually increased until, by the 13th of March, she was taking thirty-six grains daily. She was then so nearly free from spasm that she was permitted to carry plates and cups and saucers about the ward. In the evening her bowels became relaxed, she was sick, and began to complain of headache. These symptoms continued the next day, the motions being pale in colour, and slimy in appearance; the pupils, however, were not much dilated, there was no indistinctness of vision, and the tongue remained clean and moist. Nothing more was done than to omit the medicine, and the next day the bowels were quiet, the motions natural, and she felt much better. There was no return of choreic symptoms, and she left the hospital quite well on the 23d of March.

Case 11.—Catherine M'N—, æt. 10, was admitted into Hollond Ward, February 23d, 1859. She had been suffering from chorea one month, the spasms affecting the face and all the extremities. She could scarcely articulate. The pupils were large. The heart's sounds clear. The following were the quantities of belladonna taken, viz.—

Feb. 23d	— 4 grs. of the extract.	Mar. 8th	—40 grs. of the extract.
„ 24th	— 6 „	„ 9th	—44 „
„ 25th	— 9 „	„ 10th	—48 „
„ 26th	—12 „	„ 11th	—52 „
„ 27th	—12 „	„ 12th	—56 „
„ 28th	—16 „	„ 13th	—60 „
Mar. 1st	—20 „	„ 14th	—64 „
„ 2d	—24 „	„ 15th	—64 „
„ 3d	—24 „	„ 16th	—64 „
„ 4th	—28 „	„ 17th	—64 „
„ 5th	—32 „	„ 18th	—68 „
„ 6th	—32 „	„ 19th	—70 „
„ 7th	—36 „	„ 20th	—70 „

Thus, up to this date, or, in other words, within twenty-six days, she had taken 1019 grains, or rather more than two ounces of the extract of belladonna; meanwhile her appetite had remained good, and the tongue moist and clean, but red. After the first four days she was always up and playing about the ward during the day, and at night she slept soundly, but not heavily. The pupils were always large, and somewhat dilated. She never experienced any vertigo, and on one or two occasions only, the slightest indistinctness of vision. The urine, from the first, was loaded with lithates, and was of very high specific gravity, usually about 1030; and, during the last six days of her taking the belladonna it was exceedingly scanty; so much so, indeed, that not more than seven ounces were passed each day, exclusive of what was passed at stool once daily. Its sp. gr., from the 12th to the 20th of March, ranged from 1033 to 1036. She was a long time in passing it, and seemed to experience some difficulty—not pain—in doing so. In this case the spasms were almost as severe on the 20th of March as on the day of her admission, and, therefore, although no ill effects were produced by the belladonna, its use was discontinued. After she had remained five days without medicine, the administration of sulphate of zinc in one-grain doses was commenced. The choreic symptoms at once began to subside, and as the dose of zinc was increased, which it was by the 8th of April, up to

fourteen grains four times a day, they entirely disappeared. She left the hospital in good health on the 13th of April.

CASE 12. — Mary Ann S—, æt 14, was admitted into Pepys' Ward on March 2d, 1859. She had experienced repeated attacks of chorea during the last eight years, and had been thrice under treatment for it in St. George's Hospital. The present attack commenced about a fortnight ago, and the symptoms had been increasing gradually up to the date of her admission. The spasms were severe, so that she fell down yesterday, and cut her lip and bruised her face. Pupils large; heart's sounds clear. I determined to give atropine in this case, and judging from the observed effect of the extract of belladonna, I prescribed it in large doses. The following is the quantity taken each day, viz.—

Mar. 2d	—Atropine	gr. $\frac{1}{4}$	Mar. 11th	—Atropine	gr. $2\frac{1}{4}$
„ 3d	„	gr. $\frac{1}{3}$	„ 12th	„	gr. $2\frac{1}{3}$
„ 4th	„	gr. $\frac{1}{2}$	„ 13th	„	gr. $2\frac{1}{2}$
„ 5th	„	gr. 1	„ 14th	„	gr. $2\frac{3}{4}$
„ 6th	„	gr. $1\frac{1}{4}$	„ 15th	„	gr. 3
„ 7th	„	gr. $1\frac{1}{3}$	„ 16th	„	gr. $3\frac{1}{4}$
„ 8th	„	gr. $1\frac{1}{2}$	„ 17th	„	gr. $3\frac{1}{3}$
„ 9th	„	gr. $1\frac{3}{4}$	„ 18th	„	gr. $3\frac{1}{2}$
„ 10th	„	gr. 2	„ 19th	„	gr. $3\frac{1}{3}$

Thus, then, up to this date, or, in other words, within the space of eighteen days, she had taken thirty-seven grains of atropine. During the first three days the spasms were very violent, so that she had to be tied in bed, and during the first three nights she was more or less delirious; but as the tongue was moist, as the pupils were not much dilated, as there was not any indistinctness of vision, and as she was perfectly collected and sensible during the day, there was no reason for attributing the symptom to the action of the atropine. After the third night there was no return of delirium, and by the 8th of March the spasms had so far subsided as to enable her to be dressed and up about the ward during the day. The tongue had remained moist and clean throughout the attack, and the pupils, though

large, were seldom much dilated except for a short time after each dose of the medicine. She slept soundly, but not heavily. On the 19th, the last day on which the atropine was taken, my clinical assistant, Mr. Bright, reports, "She is much steadier, the pupils are of their natural size, there is no indistinctness of vision, the tongue is clean and moist, the urine clear, and the appetite good." In short there was no indication of her being affected by the drug, but as for some days it had not appeared to lessen the choreic movements its use was discontinued. She then remained without medicine until the 26th, when one-grain doses of sulphate of zinc were ordered to be taken in solution every six hours. The quantity of zinc was rapidly increased, until on the 16th of April she was taking twenty grains four times daily. The spasms gradually subsided, and she left the hospital on the 20th of April. On the 16th of March, whilst taking the large doses of atropine, she began to suffer severely from toothache, which was not relieved until the 19th instant, when the gums and face began to swell.

The following circumstances deserve to be noted. In the 1st place, feverish heat was not even temporarily produced in any instance by the administration of the medicine.

2dly. The patients were pale whilst under the influence of the drug, and in no case was any rash or erythematous blush observed on the skin, though it was looked for daily.

3dly. There was great weakness of the pulse in all the cases, and, in some, considerable quickness.

4thly. The drug did not, in any instance, exert a constipating effect; on the contrary, it appeared to prove aperient. An occasional purge was required only in Cases 1, 5, and 7.

5thly. In Cases 2, 3, 5, 7, and 10, sickness and diarrhœa were ultimately produced; but in every instance, save one, the choreic spasms had almost wholly ceased, and in the exceptional case alluded to (Case 7), had greatly subsided, before these symptoms manifested themselves. Whenever

bowel symptoms occurred, mere omission of the medicine sufficed to cause their cessation. Did the existence of spasm counteract the influence of the drug, and prevent their occurrence?

6thly. The urine was generally clear and acid, but scanty, and of high specific gravity, varying from 1024 to 1036. In Cases 3, 6, and 8, it frequently contained a copious deposit of crystallized lithic acid. In Cases 5, 7, and 9, it was usually loaded with lithates. In Case 2, for the space of a few hours, whilst the patient was under the toxical influence of the drug, it became ammoniacal almost as soon as voided.

7thly. In Case 11 some difficulty was experienced in voiding the urine throughout the period of the administration of belladonna, but this was not observed in any other cases. This difficulty passed off when the belladonna was omitted.

8thly. The tongue was always moist, but unusually red, whilst the larger doses of belladonna were being taken, and the redness passed off when the drug was omitted.

9thly. Dilatation of the pupil was very uncertain. In almost every instance the pupils were large before the administration of the medicine was commenced, and they invariably became dilated soon after a dose of the medicine was taken. The dilatation, however, was not to the degree observed when a solution of belladonna is dropped into the eye, and in most of the cases it passed off before another dose of the medicine was due. Its ordinary duration was about two hours and a half. In Case 2 excessive dilatation occurred for some hours coincidently with the occurrence of sickness and purging. In Cases 3 and 11 considerable dilatation was pretty constant. In Case 12 the dilatation was seldom great.

10thly. In two instances only (Cases 5 and 11), did the slightest indistinctness of vision occur. In Case 11 it was observed only on three occasions, and then to a slight degree, and was not accompanied by dryness of the throat, headache, or any impairment of the mental faculties. In

Case 5 it took place more frequently, and strange to say, was most complained of when the pupils were of their natural size, and were contracting freely under the stimulus of light. It was not attended by delirium or by any other indication of the action of belladonna, and the administration of an additional quantity of the drug was almost invariably followed by its removal.

11thly. The drug did not, in any case, produce the slightest narcotic effect, and, in Case 12, it failed utterly as an anodyne.

12thly. In no instance was there any evidence of its accumulation in the system.

13thly. The tolerance of the drug was not in proportion to the severity of the choreic spasms. In Case 2, in which fourteen grains of the extract daily occasioned sickness and purging, the spasms were more severe than in Case 11, in which seventy grains were given daily without disturbance of the stomach and bowels.

14thly. The curative effect of the drug was very uncertain. In Cases 1, 2, 3, 4, 6, 7, 8, and 10, its action appeared to be curative; but, in Cases 9 and 11 it failed to exercise the slightest control over the spasms, and in Cases 5, 7, and 12, it is doubtful whether the improvement ought to be attributed to its action.

Being desirous of ascertaining whether the tolerance of the drug was due to its decomposition in the stomach, or to its non-absorption, I submitted some of the urine voided by Catherine M'N—, to my friends Dr. Marcet and Mr. Kesteven for examination. The former extracted atropine enough from three ounces of the urine to kill two white mice, and narcotise several others. The latter, from two ounces of the urine, obtained sufficient to produce dilatation of a cat's eye, to afford the beautiful filamentous crystals of atropine now laid before the Society, and to give the reactions which atropine yields with iodine water, tannic acid, chloride of gold, sulphuric acid, and bichromate of potash.

The patients being all female children, I was unable to

obtain any fæces unmixed with urine, but Dr. Marcet, who kindly undertook to analyse an admixture of fæces and urine, did not obtain a larger quantity of atropine from it than he did from the urine alone. The urine and fæces were voided at the time when the patient was taking sixty-four grains of the extract of belladonna daily, and therefore, as the drug was not found in great excess in the fæces, it seems fair to conclude, that a large proportion of it had been absorbed into the blood. At all events, its discovery in large quantities in the urine proves that its want of action is not attributable to its non-absorption.

In order to ascertain whether the extraordinary tolerance of belladonna exhibited by these patients arose from their having become habituated to the use of the drug, and to a consequently induced insensibility to its action, a solution of atropine was dropped into the eye of a girl (Case 12), who, at the time, was taking three grains and a quarter of atropine daily, but whose pupils at the time were of medium size. Dilatation of the pupil ensued as under ordinary circumstances, but was of very temporary duration.

Thus, then, up to this point, five facts appeared proved—1st. That in cases of chorea extraordinarily large doses of belladonna and atropine are tolerated. 2dly. That the drug is absorbed into the blood; and therefore, that the tolerance of it is not attributable to its non-absorption, nor to its being decomposed in the stomach. 3dly. That it does not accumulate in the blood, but passes out of the system with the urine and fæces, and probably with the other excretions. 4thly. That it does not exercise that amount of control over the choreic spasms which would have been expected, from the readiness with which it is tolerated by the system. 5thly. That the tolerance of the remedy is not in proportion to the severity of the choreic symptoms.

Another question, therefore, arose, viz., whether the existence of chorea had any part in producing tolerance of the drug, or, whether that tolerance may not have been due to the age of the patients, or to some other circumstance.

Case 13.—With the view of determining this point, I gave a child, Mary Ann F—, æt. 10, in Queen's Ward, convalescent from acute rheumatism, a quarter of a grain of the extract of belladonna, in four equal doses, and increased it day by day, until, at the expiration of thirteen days, she was taking twenty-eight grains of the extract daily. When she commenced taking the belladonna she was quite free from rheumatism, and remained so until the date of her discharge from the hospital. On the first and second days of its administration dilatation of the pupil and some degree of furring of the tongue were observed; but these symptoms had passed off before the fourth day of its administration. On the tenth day, when she was taking twenty grains of the extract daily, I find a note by Mr. Bright, my clinical assistant, to the effect, that "she is quite uninfluenced by the medicine;" and on the 29th day of March, the day preceding her discharge from the hospital, at the time when she was taking twenty-eight grains daily, my clinical assistant, Mr. Leighton, made a note as follows: "Pupils natural and of medium size; no indistinctness of vision; tongue clean and moist; no dryness of throat; appetite good."

Case 14.—To another child, Elizabeth O—, æt. 7, in Crayle Ward, convalescent from scarlatina, I gave, on the 28th of March, half a grain of the extract, and increased the dose gradually, until, on the 5th of April, she took thirteen grains of the extract daily. The tongue remained moist and the appetite good throughout; there was no vertigo or indistinctness of vision, and no dryness of the throat. On the 5th of April, the day preceding her discharge from the hospital, there was not any evidence of her being affected by the drug, except slight dilatation of the pupils. She slept soundly but not heavily.

With the view of having the matter tested with children on a larger scale than is possible at St. George's Hospital, I requested my friend Mr. ——, who is attached to a large

public institution for children, to administer it cautiously and favour me with a report of the results. Accordingly, on the 24th of March he gave eleven children, varying in age from three to six, one eighth of a grain of the extract in solution three times daily. To four other children, from eight to twelve years of age, he gave, at the same time, one quarter of a grain of the extract, and increased the dose up to one grain, three times a day. These children were all in good health; the dose of the medicine was gradually increased, and the only effect observed was dilatation of the pupil, which continued for about two hours after each dose of the medicine was taken. On the 30th many of the children left the institution, and the experiment was discontinued.

On the 5th of April he gave seven children, from five to seven years of age, one third of a grain of the extract of belladonna twice a day, and continued it until the 7th, without perceiving any effect from its administration beyond that of slight dilatation of the pupil, as on the former occasion. On the 7th he prescribed two thirds of a grain to be taken twice a day, but the nurse by mistake gave a quantity of the mixture equal to a grain and a third of the extract at a dose. The result was that the children were all seized with sickness and vomiting, some of them had diarrhœa, and one of them had the violent uncontrollable delirium characteristic of belladonna. Stimulants were at once administered, the belladonna was omitted, and on the following day the toxical effects of the drug had passed off and the children were perfectly well.

On adults the medicine acts much more powerfully than on children, and a tolerance of it is not easily established. An opportunity has not yet occurred to me of administering it in full doses to patients between the ages of nineteen and thirty, but in the few persons between the ages of thirty and sixty to whom I have given it, one or two grains daily have produced vertigo or dryness of the throat and fauces, and in one instance only have I been able to increase the dose beyond four grains daily. The instance alluded to

was that of a nervous woman, aged thirty, a patient in Hollond Ward, suffering from hysterical contraction of the left arm; and in her case the dose was gradually increased up to ten grains daily. This quantity, however, occasioned so much vertigo, loss of appetite, and dryness of the throat and fauces, that I was soon obliged to discontinue its administration.

Thus, then, it would appear—1st, that the tolerance of belladonna is not attributable to the counteracting influence of choreic spasms, but is in some way connected with the age of the patient; 2dly, that a much larger dose than is usually prescribed is well borne from the first by children of tender years; 3dly, that in children, though not so in adults, a tolerance of the remedy is speedily established, so that the dose may be safely increased rapidly, but gradually; 4thly, that special care should be taken in apportioning the dose to the age of the patient and in not increasing the dose too rapidly, inasmuch as the usual toxical effects of the drug will be produced if too large a dose be given before a tolerance of the drug has been established; 5thly, that the milder toxical effects produced by the drug are of little importance, and subside without remedies as soon as the administration of the medicine is discontinued; 6thly, that adults cannot tolerate the doses of the drug which can be taken with impunity by children.

If I were to state the opinion I have formed from a close and careful observation of the cases recorded in this paper, coupled with the information supplied by Mr. —, I should say that the commencing daily quantity of the extract for a child—

Between the ages of	3	and	5	ought not to exceed one third of a grain,
„	5	„	8	„ one half „
„	8	„	10	„ two thirds „
„	10	„	15	„ one grain;

but that in each instance this dose may be safely given, and may be increased on alternate days, by an equal amount, until loss of appetite or some symptoms of the action of the

drug, other than moderate dilatation of the pupil, begin to manifest themselves. In hospital practice, where the patients are seen by a medical man three or four times daily, and in the intervals are under the eye of an experienced nurse, considerably larger doses may be given without the slightest risk. In every instance, however, the daily quantity ought to be given in at least four equal doses, for the effects of the belladonna pass off so rapidly, as evidenced by the cessation of dilatation of the pupil, that much larger doses may be thus administered daily, than it would be safe to exhibit at a single dose, or even in two or three doses.

The extraordinary difference in the tolerance of the drug observed at different periods of life is probably explicable by the medicine passing off with the urine, as also with the other excretions, more rapidly in childhood than it does in adult life.

Several questions of practical importance arise in connexion with this inquiry: 1st. May not full doses of belladonna be exhibited with advantage in hooping-cough? It is admitted that the drug is often productive of signal benefit even in the minute doses in which it has been hitherto prescribed, and it seems fair to conclude that a corresponding increase of benefit would result from the administration of larger doses. 2dly. In epilepsy, laryngysmus stridulus, and other spasmodic affections, full doses of the extract may possibly prove serviceable. It is obvious that they may be taken with impunity if due caution be observed in their administration, and they certainly deserve a trial. 3dly. May not the drug, if perseveringly employed in full doses, be productive of relief in certain forms of dyspepsia connected with infra-mammary pain, flatus, and spasms in the abdomen? Combining, as it does, antispasmodic, sedative, and slightly purgative properties, it seems likely to be useful in many such cases; and although at present my experience is too limited to warrant an opinion on the subject, I may state that I have found it give relief to two patients who had abnormally clean tongues, but were suffering from an irritable state of the stomach and of the

nerves connected therewith. 4thly. It exercises a remarkable power in controlling seminal emissions and incontinence of urine. This I can attest from oft-repeated observation, and as the experiments already detailed prove that the drug is excreted with the urine, the question arises whether, in the cases alluded to, its curative action may not be due in great measure to its topical effect, and if so, whether it might not be applied locally with advantage?

In conclusion, I would add that my best thanks are due to my colleagues at St. George's Hospital, and to other of my friends, for kindly furnishing me with cases of chorea, and for offering many valuable suggestions in aid of my investigations.

ON THE

REPARATIVE PROCESS

IN

HUMAN TENDONS

AFTER SUBCUTANEOUS DIVISION FOR THE CURE OF DEFORMITIES,

ILLUSTRATED BY A SERIES OF SPECIMENS AND DRAWINGS FROM FIFTEEN POST-MORTEM EXAMINATIONS.

BY

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The reparative process in human tendons after subcutaneous division has not, so far as I am aware, hitherto been investigated in its different stages; obviously from the following causes.

1st. A connected account of the process, from its earliest stages up to its completion, could only be based upon the examination of a large number of specimens; and,

2dly. The opportunities for pursuing this investigation must necessarily be extremely rare; since only those cases would be available for the purpose in which death occurred during the reparative process, from accidental circumstances, or from causes altogether unconnected with the operation, and also those cases in which amputation should be rendered necessary, as in one of the present instances, from

some cause also unconnected with the operation of tenotomy. The cases in which death has followed, or amputation been required, from extensive suppuration, sloughing, aneurism, &c., following tenotomy (and such cases have occurred, though very rarely), would be utterly useless for the purpose of this investigation, in consequence of the interference with the healthy process of reparation.

In the absence of observations on the human subject, the reparative process in tendons after division by subcutaneous and open wound has been carefully investigated by experiments on animals, performed by several distinguished Continental and English observers, more especially by Hunter[1] (1767?); Mayo[2] (1827); Von Ammon[3] (1837); Pirogoff[4] (1840); Paget[5] (1849); Gerstaecker[6] (1851); Thierfelder[7] (1852); and Boner[8] (1854).

The conclusions arrived at, however, by the above-mentioned authorities as to the nature of the reparative process,

[1] Probably about this date. In Ottley's life of Hunter (see 'The Works of John Hunter,' Palmer's edition, London, 1837) it is stated that, in the year 1767, Hunter ruptured his tendo Achillis whilst dancing, and that this accident led him to examine into the process by which divided tendons are reunited. His experiments on dogs by subcutaneous division of the tendons are then referred to. The Hunterian preparations in the College of Surgeons are from the ass and deer.

[2] 'Outlines of Pathology,' London, 1827, and the three subsequent editions, in which the same account is repeated.

[3] 'De Tenotomiæ Physiologia, Experimentis Illustrata.' Dresdæ, 1837.

[4] 'Ueber die Durchschneidung der Achillessehne, als operativ-orthopädisches Heilmittel von Nikolaus Pirogoff. Mit sieben Tafeln.' Dorpat, 1840. For an account of these experiments see also Gerstaecker's 'Diss.'

[5] 'Med. Times and Gazette' for 1849; and also 'Lectures on Surgical Pathology,' vol. i, London, 1853.

[6] 'Dissertatio de Regeneratione Tendinum post Tenotomiam,' Adolphus Gerstaecker. Berolini, 1851.

[7] 'Diss. Histol. De Regeneratione Tendinum, F. F. Thierfelder, Micensæ, 1852.

[8] 'Die Regeneration der Sehnen,' J. H. Boner; 'Archiv für Pathologische Anatomie und Physiologie und für Klinische Medicin, R. Virchow. Berlin, 1854, p. 162.

and especially in reference to the influence of extravasated blood, inflammatory lymph, and the sheath of the tendon, were so widely different, that in the year 1855 I re-investigated the subject, in a series of experiments performed on sixteen rabbits; the parts removed from fourteen of which, illustrating the process from the second to the sixty-second day, were exhibited to the Pathological Society, and the results were published in the sixth volume of this Society's 'Transactions.'[1] Two other animals lived to the periods of six months, and a year.

I will here only observe, that my investigations generally confirmed the account of the process previously given by Mr. Paget, especially with regard to the influence of blood and the inflammatory process in interfering with, rather than assisting, the reparative effort; in this respect, therefore, they were at variance with the opinions of all the other authorities above mentioned. My investigations also confirmed Mr. Paget's observation, with regard to the development of the new connective tissue from a proper reparative material effused for this purpose, and described by Mr. Paget as "nucleated blastema," a material in which the cell-forms do not advance beyond the condition of nuclei, and thus differ from inflammatory lymph, in which fibro-cellular tissue is developed from nucleated cells. The most material point in which my observations differed from those of Mr. Paget was in reference to the influence of the sheath of the tendon, which I found not divided, and generally but very little injured, in the subcutaneous operations. It therefore appeared to me, that in the reparative process the sheath was of primary importance in maintaining a direct connexion between the divided extremities of the tendon. In my experiments, also, it appeared that the new reparative material was infiltrated between the fibrous elements of the sheath, which, therefore, at once formed the matrix for the newly formed tendon, and also determined its direction and definite form, as described by Thierfelder.

[1] 'Trans. Path. Soc. of London,' vol. vi, 1855.

Mr. Paget, on the other hand, completely ignores the influence of the sheath in the reparative process, and after describing the general infiltration and succulency of all the tissues surrounding the tendon, and the subsequent changes which occur in any inflammatory lymph and blood which may be effused, and the manner in which the development of the nucleated blastema laid down between the separated extremities of the divided tendon appears to proceed, observes that "a single well-designed and cord-like bond of union is thus gradually formed, where at first there had been an uniform, and seemingly purposeless, infiltration of the whole space left by the retraction of the tendon."[1] Mr. Paget also states that, in experiments in which the tendon was divided by an open wound, when the wound through the integuments healed quickly, the case proceeded like one in which the subcutaneous division had been made, and therefore he adduces this in proof "that it is unimportant for the healing of divided Achilles tendons, whether the cellular sheath or covering of the tendon be divided or not." The facts and observations which have led me to a different conclusion on this point, will be specially adverted to in the general description of the reparative process given below.

Valuable as such experiments performed on animals undoubtedly have been, the necessity of investigating the same process in the human subject still remained, and a connexion of more than eight years with the Royal Orthopædic Hospital has afforded me the opportunity of examining *post mortem* the reparative process in human tendons after subcutaneous division, in nine patients who had been operated upon by myself, one by Mr. Tamplin, and two by my late respected friend and colleague, Mr. Lonsdale. I have also examined after death one of my own cases in private practice. These patients all died from various causes unconnected with the operation, at periods varying from four days to three years after the division of the

[1] Op. cit., p. 269.

tendons. I am also indebted to Mr. Erichsen and Mr. Curling for the opportunity of examining two valuable cases, one after death, and the other after amputation. The details of Mr. Erichsen's case have already been published, together with a lithographed plate of the appearances presented, in the 'Transactions of the Pathological Society.'[1] I have therefore examined, in the recent state, fifteen cases in which subcutaneous tenotomy had been performed, at periods varying from four days to three years previously, and from twelve of these either the specimens or drawings —in ten cases the specimens—are presented to the Society. I have also examined several preparations preserved in spirits, in the possession of Mr. Tamplin and the late Mr. Lonsdale. In most of the cases above referred to, several tendons had been divided at different dates, thus multiplying the stages at which I have been enabled to investigate the reparative process; I have now, therefore, ventured to submit the results of my observations to the Fellows of this Society.

Cases and Post-mortem Examinations described in Abstract.

Case 1.—*Four days after operation.*—Child, æt. 4 weeks. Severe congenital talipes varus. Tendo Achillis and tibialis anticus divided. Death from pneumonia.

Post-mortem examination.—Divided extremities of tendo Achillis three quarters of an inch apart, but connected by the cellular sheath of the tendon, which was vascular and succulent, and enclosed a small quantity of extravasated blood. Divided extremities of the tendon square, abrupt, and unaltered in appearance and texture.

Appearances of divided anterior tibrial tendon similar, but the connexion by the cellular sheath less distinct, and separation greater—one inch.

Case 2.—*Eleven days after operation.*—Child, æt. 8 weeks. Severe congenital talipes varus. Tendo Achillis,

[1] 'Trans. Path. Soc. of London,' vol. viii, London, 1857.

tibiales anticus and posticus, and flexor longus digitorum tendons divided. Death from diarrhœa.

Post-mortem examination.—Divided extremities of tendo Achillis seven eighths of an inch apart, but connected by a solid bond of union, of a soft and somewhat gelatinous consistence and blood-red colour; general vascularity. Divided extremities of the tendon slightly rounded, and a little softer than natural, but retaining their dead-white colour, and pretty firmly connected with the uniting bond of new material.

Tibialis posticus tendon.—Divided extremities seven eighths of an inch apart, and without any direct connexion. Connected with each of these divided extremities of the tendon was a portion of new material, a quarter of an inch in length, of firm consistence, translucent in appearance, and of a ruddy tinge. It seemed doubtful whether union would ever have taken place between these divided extremities, their permanent connexion with the sheath behind the malleolus, as in Case 12, appearing more probable.

Flexor longus digitorum.—Divided extremities half an inch apart, but directly connected by a thin band of fibrous tissue.

Microscopical examination.—The new connective tissue, formed between the divided extremities of the tendo Achillis, consisted of nucleated blastematous material, in which, after the addition of acetic acid, numerous small oval and rounded nuclei were apparent, irregularly scattered, and not arranged in linear series. The connective tissue had no disposition to split into fibres, but, whether teased with needles, or compressed with the glass, resembled at its edges torn membranous material. The portions of new tissue connected with the extremities of the posterior tibial tendon presented similar appearances, but with less intermixture of inflammatory effusion, and a much smaller quantity of molecular oil.

Case 3.—*Sixteen, twenty-three, and thirty days after operation.*—Child, æt. 3 weeks. Severe congenital varus of both feet. The tibial tendons of both feet, and the tendo

Achillis of the right foot, were divided. Death from pneumonia.

Tendo Achillis, sixteen days after operation.—Divided extremities rather more than half an inch apart, but connected by a tough bond of new material, of a ruddy, blood-stained colour, equal in diameter and bulk to the tendon it served to connect. The divided extremities of the old tendon retained much of their abrupt and square outline, the margins only being a little rounded. They appeared not to have undergone any material change, though they were firmly connected with the new tissue by a very fine dovetailing arrangement, scarcely traceable by the naked eye.

Microscopically examined, the connective tissue between the divided extremities of the tendo Achillis appeared to consist of old fibro-cellular tissue, such as probably formed the sheath or fibro-cellular investment of the old tendon, intermixed with a large quantity of granular, nebulous material, studded with molecules and larger globules of oil, the latter probably derived from included portions of fat. After the addition of acetic acid, numerous small rounded and oval nuclei were brought into view. These nuclei had no disposition to a linear arrangement. At the junction of the new with the old tissue, a coarse separation of the structure of the old tendon into obtusely rounded bundles of fibres, between which the new material was inserted, was very evident; and the new tissue at this part was much clearer than towards its central portion, and, after the addition of acetic acid, was seen to be thickly studded with small oval and rounded nuclei, without any disposition to a linear arrangement.

Tibiales anticus and posticus tendons of left foot, twenty-three days after division.—Divided extremities of anterior tibial tendon three quarters of an inch apart, but connected by a firm cylindrical bond of new material, easily distinguished, by its ruddy tinge and appearance of vascularity, from the old tendon.

Divided extremities of posterior tibial tendon half an inch apart, but connected by a tough bond of new material, as

in the anterior tibial. The extremities of the old tendon retained their abrupt and square outline, but slightly rounded at the margin, without any bulbous enlargement or dovetailing visible to the naked eye.

Microscopically examined, the new material approached in its structural arrangement the characters of well-formed tendon sufficiently to justify its being described as newly formed tendinous structure. It had a distinctly fibrillated character, though pale and delicate when compared with old tendon, but yet its margins would not split or fray out into separable fibres or bundles of fibres. The addition of acetic acid exhibited an appearance of elongated nuclei, united by filaments in a linear arrangement, and running in parallel lines, with almost as much regularity as in the old tendon; but the nuclei, if they may be so considered, in the new tendon were dotted and more elongated than in the old tendon, in which the nuclei were clear and well defined. At the junction of the new with the old tendon, the structure was more translucent than at any other part, and there was an absence of the linear arrangement of elongated nuclei and of the fibrillated appearance of the new tissue; at this part only the smallest nuclei, generally of spherical or oval form, were clustered without any regularity. Numerous vessels existed in the new tendon, the larger trunks running transversely or obliquely across it, as if formed from the circumference towards the centre, and then dividing; the smaller branches were either continued obliquely, or ran in a longitudinal direction.

Tibiales anticus and posticus tendons of right foot, thirty days after division.—Divided extremities of anterior tibial tendon nearly three quarters of an inch apart, but connected by a firm bond of new material like that in the corresponding tendon of the opposite leg.

The posterior tibial tendon, a little above the inner malleolus, presented externally a ruddy tinge from blood-staining and vascularity of adjacent areolar tissue, resembling in some degree the appearances of the corresponding tendon of the opposite leg, but a longitudinal section and minute

examination showed that this tendon had not been divided. This specimen might have been considered by some as exhibiting a very perfect condition of repair, and as an example of a linear cicatrix, especially as the case had proceeded as favorably as if it had been divided.

Case 4.—*Eighteen days after operation.*—Child, æt. 4 weeks. Severe congenital varus of right foot. Tibialis posticus and flexor longus digitorum divided. As it was supposed that the posterior tibial artery had been wounded, the other tendons were not divided. Death from diarrhœa. Instead of gradual extension being made, the foot was retained in the deformed position by a tin-splint, and pressure applied—the plan we adopt when the artery is supposed to have been wounded. This accounts for the unusually slight extent of the separation of the divided extremities of the posterior tibial tendon. The specimen, which is now in St. Bartholomew's Museum, was carefully examined by Mr. Paget, and the following is an abstract of the description written by him.

Divided extremities of *posterior tibial tendon* only one line apart; upper extremity grayish-looking, succulent, slightly swollen, and rounded or bulbous; lower extremity unchanged in texture, and free end rectilinear. Between the two, and attached especially to the end of the upper portion, was a firm, tough, elastic, grayish substance, apparently a connecting bond of reparative material, about a line in length, and less than half a line in width or thickness; smaller, therefore, than either portion of the tendon. This substance was united to the adjacent tissues, yet could be dissected from them. Portions of it, examined with a microscope, appeared to be composed of reparative material placed among the filaments of fibro-cellular tissue, such as might have collapsed into the space between the separated ends of the tendon. It contained abundant blood-vessels, but was essentially composed of compact, closely-wrinkled tissue, like that of new tendon in the rabbit, dotted with minute molecular matter. The addition of acetic acid made

it quite clear, and showed in it a few nuclei, besides those of the blood-vessels that traversed it.

The tendon of the flexor longus digitorum was divided somewhat obliquely. Both its portions were very slender, and the upper one was so wanting in firmness that it was difficult to trace its end; neither did there appear any distinct new-formed bond of union between them; they were held together by white tissue, apparently the fibro-cellular, which had invested the tendon, and to which the portions of the tibialis posticus and their uniting medium were adherent.

Case 5.—*Five weeks and six weeks after operation.*—Child, æt. 6 weeks. Congenital talipes varus, not severe. Tibiales anticus and posticus, and flexor longus digitorum, divided six weeks, and the tendo Achillis five weeks, before death, which resulted from acute pneumonia.

Tibialis anticus.—Divided extremities nearly half an inch apart, but connected by a tough bond of new material, equal in thickness to the extremities of the tendon, and similar in appearance to the other specimens about this date.

Tibialis posticus and flexor longus tendons.—Divided extremities rather less than a quarter of an inch apart, but connected by new material of a firm, compact texture, and pale colour; grayish, with a light-ruddy tinge from vascularity. Both these tendons adherent to each other and to the adjacent bone-surface. The less separation of the tendons in this instance is explained by the case not being of a severe form.

Tendo Achillis.—Divided extremities a quarter of an inch apart, but firmly connected by a tough bond of new material, presenting on section a translucent appearance, and light-ruddy tinge from vascularity, readily distinguished from the opaque-white extremities of the old tendon. The junction of the old with the new tendon was indicated by a fine dovetailing process.

Microscopically examined, the new material exhibited a delicate, fibrillated appearance, but would not split or fray out into separable fibres. The addition of acetic

acid brought into view abundant nuclei, and an arrangement in parallel linear series was evident, so that a perfecting of the new connective tissue towards the characters of well-formed tendon was clearly traceable.

Case 6.—*Six weeks after operation.*—Child, æt. 9 weeks. Severe congenital talipes varus. Tibiales anticus and posticus, and flexor longus digitorum, and tendo Achillis, divided at the same time by Mr. Tamplin, in whose possession the specimen now is. The post-mortem examination was made by me, and the specimen minutely examined at the time, and drawing made. Cause of death not recorded. Several muscular irregularities, with additional tendinous slips, passing between and connecting the various tendons around the ankle-joint and in the foot, existed in this case.

Tibialis posticus and flexor longus digitorum.—Divided extremities a quarter of an inch apart (or rather more), but connected by a tough bond of new material, of a grayish appearance, and with some vascularity. Both tendons were inseparably fused in this material, which was also closely and firmly adherent to the surface of the bone.

Tibialis anticus.—Divided extremities half an inch apart, but in consequence of the splitting of this tendon, and its connexion with the anterior annular ligament, its cylindrical form and definition were less distinct than usual. The connecting bond appeared to consist of old fibrous tissue, thickened by infiltration of new reparative material.

Tendo Achillis.—Divided extremities rather less than half an inch apart. The depression of the heel did not appear to be complete in this case. On section, the new material presented a grayish translucent appearance, so that it was readily distinguishable from the old tendon; its vascularity was evident. At the junction of the new with the old tendon the dovetailing process was very obvious, especially at the lower extremity, where this appearance was coarse, and the old tendon presented a slightly bulbous enlargement.

Microscopically examined, the new material in the several

tendons presented a delicate, fibrillated appearance, but would not fray out into separable fibres, except in some portions from the tendo Achillis, and in this situation some bands of old fibro-cellular tissue, blended with the new material, were probably examined. In all the tendons acetic acid, applied to the new material, brought into view a parallel arrangement of elongated nuclei, and at the junction of the old with the new tendinous material the latter was clearer than in its central portion, and studded with very small oval or rounded nuclei, irregularly scattered over the field.

Case 7.—*Three months after operation.*—Girl, æt. 8 years. Talipes equinus of right foot, associated with partial paralysis of the muscles of the leg. Tendo Achillis divided. Death from sloughing sore-throat; scarlatina? Post-mortem examination made by Mr. Coghlan, of Notting Hill, and myself. The whole of the spinal cord was examined, with a view to the pathology of infantile paralysis, but no abnormal condition was detected in any part. All the muscles of the leg below the knee were in a more or less advanced state of fatty degeneration, but the muscular structure was not so completely destroyed as I have seen it in other cases. The form of the foot was natural, and without excessive depression of the os calcis.

Tendo Achillis.—The cylindrical form of the tendon was well defined on its superficial and lateral aspects, but the definition of its deep surface was obscured by adhesions. At the seat of division its surface was not so smooth and glistening as above and below this portion; from this part no loose, separable sheath could be dissected. A slight increase of vascularity also identified the seat of operation. On section, the divided extremities of the tendon were seen to be three quarters of an inch apart, but firmly connected by a tough and close-textured bond of new material, equal in bulk and thickness to the tendon it served to unite, and of a slightly striated or fibrous appearance, which was added to by a few injected vessels running longitudinally

through its texture. The junction of the new with the old tendon by a dovetailing process was very clear and distinct. The cut extremities of the old tendon were slightly puckered in at the exact line of junction, especially at the upper extremity, so that the new material for about one eighth of an inch ensheathed the extremity of the old tendon, but beyond this, especially at the lower extremity, a slight bulbous enlargement of the old tendon existed.

Microscopically examined, the structure of the new material, or new tendon, so closely resembled the structure of the old tendon, that careful observation was necessary to distinguish them. The new material presented a distinctly fibrous appearance, resembling that of the old tendon, more easily splitting into separable fibres than in any specimen I have examined at an earlier date. Acetic acid rendered the tissue translucent, and brought into view numerous elongated nuclei, arranged end to end in parallel linear series, an appearance also exhibited in the old tendon after the addition of acetic acid.

The reparative process by regeneration of tendinous structure between the divided extremities of the old tendon (the portion of new tendon being three quarters of an inch in length) was beautifully illustrated by this specimen, one half of which I have presented to the Royal College of Surgeons.

Case 8.—*Three or four months after operation.*—A boy, æt. 16 years. Talipes equinus. Tendo Achillis divided by Mr. Tuson, of the Middlesex Hospital. Specimen formerly in the possession of my late colleague, Mr. Lonsdale, and now in St. Bartholomew's Hospital. Divided extremities of tendo Achillis one inch and a quarter apart, but connected by a tough bond of new material, equal in bulk to the thickness of the tendon. The dovetailing process of the new with the old tendon was obvious at the line of junction. Imbedded in the new connecting material were the remains of a rather large clot of blood, and the reparative process was imperfect at this part. The new material also presented

the appearance of a more irregular interlacement of fibres than usual. Altogether, there was more evidence of local disturbance at the seat of the operation, from extravasated blood and inflammatory effusion, than I have generally seen.

CASE 9.—*Seven months after operation.*—A boy, æt. 10 years. Congenital talipes varus. Operated upon by my late colleague, Mr. Lonsdale. Death from scarlet fever. Post-mortem examination made by me.

Tendo Achillis.—Divided extremities one inch apart, or rather more, but connected by a tough and very strong bond of new tendinous structure, equal in bulk and thickness to the extremities of the tendon it served to connect. The new material had a grayish translucent appearance, with very little evidence of vascularity to the naked eye, but with slight traces of a fibrous arrangement of its structure; some fibrous bands, approaching somewhat to the opaque pearly lustre of old tendon, passing obliquely through its substance. This appearance I have seen more marked in cases at a later date. The deep surface of the new tendon was somewhat ill defined, from its connexion with thickened bands of fibro-cellular tissue intersecting the adjacent fat.

Microscopically examined, the new material presented a distinctly fibrous character, and although not splitting easily into separable fibres, still the edges of any portion examined exhibited a tendency to such splitting, and in some parts would fray out into delicate fibres. Acetic acid rendered this structure transparent, and the arrangement of elongated nuclei in parallel linear series was distinct.

CASE 10.—*About one year after operation; and also at two subsequent periods, probably about seven and four months, but the precise dates uncertain.*—A girl, æt. 3 years. Congenital talipes varus. Operated upon by my late colleague, Mr. Lonsdale. The case was one of unusual severity, and the operation had to be repeated. Death from scarlet fever.

Tendo Achillis.—The appearances left after three divisions

of this tendon were clearly traceable. The divided extremities of the old tendon at their point of widest separation were one inch and a half apart, but within this space were two detached portions of old tendon, which together measured about half an inch in length, and these were firmly and intimately blended with the new tendinous structure, formed after the three divisions of the tendon. These detached portions of old tendon retained their opaque pearly lustre, and were thus readily distinguishable from the intermediate portions of new connective tissue, which presented a grayish, translucent appearance, as seen in all the other specimens some months after the operation. The junction of the old with the new tendon, by the process of dovetailing, was obvious, both in the detached portions, and at the extremities of the old tendon. The new tissue had but very slight traces of vascularity to the naked eye, and only slight indications of a fibrous arrangement of its structure.

Microscopically examined, the new tendinous structure had a distinctly fibrillated appearance, and, after the addition of acetic acid, exhibited the parallel linear arrangement of elongated nuclei previously described.

Case 11.—*One year and a half after operation.*—A girl, æt. 9 years. Talipes equinus, coexisting with a recurrent fibroid tumour in the calf of the leg. After an incomplete removal of the tumour, the connexions of which were found to be too deep to admit of complete enucleation, Mr. Curling divided the tendo Achillis, and cured the deformity of the foot. The tumour returned, and then Mr. Curling amputated the leg.[1] For the opportunity of examining the leg I am indebted to Mr. Curling. It is important also to mention that the deformity had returned to an extreme degree previous to the amputation.

Tendo Achillis.—After exposing this tendon by dissec-

[1] 'Path. Soc. Trans.,' London, vol. vi, p. 345; and also vol. vii, p. 345.

tion, it might be said, from a rough external examination, that no traces existed of the tendon having been divided. Its external form and size were quite natural, without any irregularity of surface, which was readily defined by dissection, nor was there any increased vascularity; but a closer examination showed that through two inches and a quarter of the length of the tendon, its surface did not present such a smooth, glistening appearance as in the normal condition, and that in this portion we could not dissect from its surface the layer of loose-textured areolar tissue, which naturally surrounds the tendo Achillis, and is described as its cellular sheath.

After making a longitudinal section of the tendon, it was at once apparent that it had been divided, and that the divided extremities were separated to the distance of two inches and a quarter, but were firmly connected by a tough bond of new connective tissue, or new tendon, equal in bulk and thickness to the tendon it served to re-unite. The new tendinous structure presented a grayish, translucent appearance, contrasting very obviously with the opaque, white, pearly lustre of the old tendon above and below it, and exhibited only slight traces of vascularity. Its structure was homogeneous rather than fibrous in appearance; it had no longitudinal fibrous arrangement, but some opaque, white, glistening fibrous bands appeared to traverse its structure obliquely, and presented on the cut surface a somewhat feathered appearance. The junction of the new with the old tendon was very distinctly indicated by the process of dovetailing described in the other specimens, the new material, which preserved its translucent appearance, being inserted between the split fibres of the old tendon, which retained their opaque pearly lustre. This was equally distinct at the upper and lower extremities of the old tendon, in neither of which was there any appearance of bulbous enlargement.

Microscopically examined, the new connective tissue, or new tendon, so closely resembled the appearances presented by the old tendon, that it would certainly have been

difficult for any one, unaccustomed to examine these structures, to point out any characters by which they might be readily distinguished. The new tendinous tissue presented very distinctly the appearance of separable fibrous structure, differing only from the old tendon in being more delicate, and less readily separable, the margins only fraying out. The addition of acetic acid did not render the new tissue so clear and translucent as in the cases at an earlier date, nor was the arrangement of elongated nuclei in parallel series so distinct. The fibrous character of the new tissue remained faintly traceable long after the addition of acetic acid, and the appearance of elongated nuclei was obscure, so that the latter might well be supposed to be replaced by dark lines or spaces. The same might be said of the old tendon after the addition of acetic acid, faint tracings of fibres and indistinct elongated nuclei taking the place of complete translucency, and distinctly elongated nuclei arranged in parallel series, as seen in the specimens of new tendon at an earlier date, and also in the original tendinous structure in infants. The new tissue was studded with a small quantity of molecular oil.

Much of the interest which attaches to this case arises, not only from the circumstance that a portion of new tendon, two inches and a quarter in length, could be proved to exist between the divided extremities of the old tendon one year and a half after its division, but that, coexisting with this additional length of new tendon, the deformity, viz., talipes equinus, had returned in an extreme degree previous to amputation. It is, therefore, proved that in this case the relapse of the deformity was not due to the contraction and absorption of the new connective tissue, but depended upon changes taking place in the muscular structure. The same fact is also illustrated by the next case, but the pathological changes in the muscular structure were of a different nature.

CASE 12.—*Two years after the last operation, but a longer, though uncertain, period after the previous operations.*

—A man, æt. 37. Non-congenital equino-varus of paralytic origin. This specimen has already been described at great length, and figured in the 'Transactions of the Pathological Society of London,' vol. viii, 1857; and to this description I must refer the reader. It is here only alluded to as one of a series, and its place indicated in reference to the period of the reparative process.

Case 13.—*Three years after operation.*—A lady, æt. 42. Equino-varus of one foot, and equino-valgus of the other, produced by infantile paralysis. Both Achilles-tendons divided. Death from phthisis.

Achilles-tendons.—The divided extremities of these tendons were found to be, one an inch and a quarter, and the other an inch and an eighth apart, but firmly connected by a strong bond of union, or new tendon, equal in bulk and thickness to the extremities of the tendons it served to unite, and presenting a grayish, translucent appearance, by which it was readily distinguishable from the old tendon. The new material had, in a more marked degree than I have seen in any other human specimen, a longitudinally striated appearance. The cut extremities of the old tendon were very distinct, from the contrast of colour above described, and the line of junction of the new with the old tendon was indicated by the dovetailing process described in the previous specimens. The new material extended at its circumference slightly beyond, and ensheathed the extremities of the old tendon.

Microscopically examined, the new connective tissue presented the appearance of well-formed tendon; and when portions of it were examined side by side with portions of the old tendon, the general resemblance was so close that, without the evidence presented by the recent section, it might very well have been said that no material differences of structure existed in any part; the presence of any new material might have been doubted, and an inference favorable to the *linear cicatrix theory* drawn. The new tendon presented a distinctly fibrous appearance, and

readily split into separable fibres, more delicate than the fibres of the old tendon, and dotted with minute molecules of oil. In the old tendon, however, molecular oil was very abundant, an appearance which I have not seen before in old tendon; and this, together with the increased separability of its fibrous structure, might, perhaps, be regarded as changes of degeneration, depending upon the long-standing paralytic affection. After the addition of acetic acid the appearances were very similar in the old and new tendon, viz., a shaded, nebulous appearance, with pale and indistinct traces of its fibrous structure, and dark crooked lines, as of the remains of elongated nuclei arranged in ill-defined parallel series.

This completes the series of thirteen cases, of the recent appearances of which, with one exception, I have been enabled to give a detailed account from my own dissection and microscopical examination; accompanied in every instance by a drawing.[1] In all these cases the reparative process has been observed in different stages between four days and three years after the subcutaneous division of the tendons.

I also possess short notes of three other post-mortem examinations, which have been made by me at periods between one and seven months after the operation; and from one of these cases I have preserved a portion of the foot, but have no detailed account of the post-mortem appearances, nor were any drawings made at the time. I am not, therefore, enabled to state the exact length of new material formed in these cases; but can, with confidence, assert that the general appearances of these three cases corresponded with the others above described of about the same periods after operation.

[1] From ten of the above cases the specimens were exhibited to the Society.

General Summary of the Reparative Process.

The principal events in this process of development of new tendon after subcutaneous division, may, I believe, be described in a general summary, as follows. Let it be understood, however, that the description especially applies to the tendo Achillis. Essentially the reparative process is no doubt the same in all tendons; but as the anatomical conditions, under which tendons are placed in some situations, vary,—for instance, where tendons pass through dense tubular sheaths, as the tibialis posticus behind the inner malleolus, the flexor tendons along the fingers, &c., there is an absence of the investing cellular sheath, and of the fat and cellular tissue which surrounds the tendo Achillis and other tendons similarly placed,—and as these conditions sometimes interfere with the perfection of the reparative process, to which I will afterwards refer, it will be better to limit the general description to the process as it appears to me to occur in the tendo Achillis, which I prefer for illustration, though it would equally apply to other tendons placed under similar anatomical conditions. The process, it appears to me, may be best described under the four following heads, under which the principal events may be most conveniently grouped. They cannot be considered as so many different stages, because two or three of the processes described are always proceeding simultaneously.

1st. *Immediate results of the operation.*—After the subcutaneous division of the tendo Achillis in the human subject, separation of the divided extremities of the tendon takes place to a very variable extent. Under ordinary circumstances the separation of a divided tendo Achillis in an infant is about half an inch, and in the adult from one to two inches. This is produced in the following way: the upper portion of the divided tendon is drawn upwards by the contraction of the muscular fibres, and the lower portion is drawn downwards, in proportion as the foot is

restored to its natural position. The separation must, therefore, depend upon the capability of the muscular fibres to contract, and also upon the flexibility of the ankle-joint; and as in deformities the muscular structures are found in all conditions, from that of health to complete degeneration, and as the ankle-joint is also found in every degree of rigidity, from the adapted shortening of the ligamentous and other structures, &c., the separation of the divided tendon will vary in a proportionate degree. The circumstances which limit the separation of the divided extremities of the tendon are—1st, degeneration of the muscular tissue, as in non-congenital cases of deformity of long standing and of paralytic origin, &c.; 2dly, rigidity of the ankle-joint, produced by a variety of causes, but in congenital cases principally by the adapted shortening of the ligamentous structures, and in the non-congenital cases by old inflammatory infiltration of the structures surrounding the joint, &c.; 3dly, old adhesions in the neighbourhood of the tendon, such as result from a previous operation, or other causes. Frequently two, and sometimes all three, of these conditions coexist, and in such cases scarcely any separation of the divided extremities of the tendon will take place at the time of the operation, so that the surgeon may be led to doubt whether the tendon has been completely divided.

Influence of the sheath.—The separated extremities of the divided tendon still remain indirectly connected with each other through the medium of the cellular sheath of the tendon, by which I mean the loose-textured areolar tissue which closely invests and surrounds the tendo Achillis, and which is never divided, and often appears to be but very little injured, in a subcutaneous operation; it evidently yields before the knife as the latter passes through the tense tendon. Even if this cellular sheath should be completely divided as in an open wound, it would not retract with the tendon, in consequence of its connexion with the subcutaneous fat and cellular tissue in the human subject; and in rabbits, in which animals no subcutaneous fat exists in the neighbourhood of the tendo Achillis, it would not retract, in

consequence of its connexion with the deep fascia and sheaths of the deeper tendons. The influence of the sheath, therefore, which I hold to be of great importance, is not destroyed, as has been supposed by Mr. Paget, by the open wound, though it is certainly impaired; and the reparative process in these cases is proportionably less perfect, but not to an extent permanently to interfere with the formation of a sufficient quantity of new tendon. (See experiments on the reparative process after division by open wound, in the sixth volume of 'Path. Soc. Trans.') This cellular investment of the tendo Achillis, which is scarcely demonstrable as a sheath in an anatomical dissection, becomes very easily demonstrable as such after the subcutaneous division of the tendon, because the loose-textured areolar tissue, of which the sheath is composed, is then put on the stretch, and being very little injured in the operation, as I have explained, from its yielding before the knife, presents the form of a tubular sheath passing between, and connecting indirectly the separated extremities of the divided tendon. This is very readily demonstrated in the rabbit (see experiments previously referred to, and the accompanying plate, in which this appearance is represented). I have also seen the same condition in the human subject.

A very small quantity of blood is generally effused at the time of the operation within the sheath, now of tubular form, and is seen adherent to the upper and lower extremities of the tendon, but principally to the upper extremity. In many of my experiments on rabbits, there was scarcely a trace of blood; and in two examinations in the human subject, a few days after the operation, I found only a very small coagulum, certainly not sufficient to take any important part by its organization (the possibility of such a process being admitted) in the formation of the large quantity of new tendon required to connect the separated extremities of the old tendon. If the effused blood should be sufficient to fill the sheath and infiltrate the surrounding tissues, the reparative process will be retarded and rendered proportionably less perfect. Such an event must be regarded as

an unfavorable accident of the operation, instead of an essential part of the process, as it has generally been regarded, especially by the French and German authorities. In such cases, the greater part of the blood will become absorbed, and the remaining and firmer portion of the clot will be found at a late period inclosed in the midst of the new tendon; this I have witnessed in several instances both in the human being and in the rabbit.

At the time of the operation, then, the separation of the divided extremities of the tendon, which remain connected by the sheath, which now assumes a tubular form, and the effusion of a small quantity of blood, are the ordinary occurrences.

2d. *Commencement and nature of the reparative process.*—The true reparative process commences in the human subject by increased vascularity of all the structures at the seat of the operation, viz., the subcutaneous cellular tissue and fat, and the cellular sheath of the tendon, now of a tubular form, passing between and connecting the separated ends of the tendon. In the rabbit the increased vascularity is confined to the connecting tubular sheath. For this reason, then, I regard the increased vascularity of the sheath in the human subject as essentially the first step in the reparative process, the extension of the vascularity to the cellular tissue and fat being the necessary consequence of the existence of this tissue, but unimportant as regards the reparative process. Increased vascularity of the sheath is followed by infiltration of a blastematous material into its meshes, or spaces, between its fibrous elements, so that the sheath, now forming the matrix in which the reparative material is effused, presents a vascular and succulent appearance. In the human subject this effusion and succulency may sometimes extend to the surrounding cellular tissue and fat, especially in clumsily performed operations, which may be followed by inflammation; but essentially I do not believe it extends beyond the sheath, because in carefully performed operations, followed by appropriate treatment, viz., a compress and bandage applied immediately after the

operation, the gap between the divided extremities of the tendon remains as a depression for several days; it does not become filled up, as it would do if the tissues were at once infiltrated; and an external examination by the finger readily detects the square and abrupt extremities of the divided tendon freely moveable in the cellular tissue, which to the touch seems flaccid rather than distended by infiltration.

Development of the reparative material.—The blastematous material infiltrated into the meshes of the sheath increases in quantity, and, microscopically examined, exhibits the development of innumerable small oval nuclei. A few cells, of large size and irregular form, with granular contents, or perhaps with one or more nuclei, and studded with minute molecules of oil,—cells such as are met with in ordinary inflammatory effusions,—may also be found according to the extent of the inflammatory lymph which may be accidentally intermixed with the nucleated blastema,—so called by Mr. Paget, and described by him as the proper reparative material as distinguished from inflammatory lymph, in which development proceeds through nucleated cells, as in the ordinary exudation from a granulating surface. A blastematous material, then, in which the cell-forms do not pass in development beyond the stage of nuclei, appears to be the proper reparative material from which new tendon is developed, and any admixture of inflammatory lymph in which the ordinary inflammatory exudation-cells are developed, must be regarded as an accidental complication of the reparative process, instead of being an essential part of it, as is generally supposed.

The changes which subsequently occur in the nucleated blastema are—1st, the formation of capillary blood-vessels. In the specimen from a child, exhibiting the reparative process on the eighteenth day after division, above described, examined microscopically by Mr. Paget and myself, newly formed and forming capillary blood-vessels with their nucleated parietes were beautifully seen after the addition of acetic acid, and were very abundant. 2dly, the nuclei assume an elongated, spindle-, or oat-shaped form, and are

seen, after the addition of acetic acid, to be arranged in parallel linear series. Whether fibres are formed in this way, or in what manner the elongated nuclei are disposed of, may still be matter of opinion; but I am inclined to believe in the development of fibres from this process, after carefully examining numerous specimens, both in my experiments in rabbits, and in the specimens from the human being.

As a gradual change towards perfecting the new connective tissue in its structural characters and general resemblance to the old tendon, the divided extremities of which it serves to connect, a fibrillated appearance of the new tissue is traceable under the microscope, and, in the course of time, a more distinctly fibrous appearance, *i. e.* tissue capable of splitting into distinct fibres under the microscope, becomes developed. The fibrous tissue, however, always remains more delicate and less distinctly separable than in old tendon.

3d. *General appearance and structure of the newly formed connective tissue, or new tendon.*—By the development of the blastematous material above described, a solid bond of union is formed between the divided extremities of the tendon, of variable length, according to the distance between the divided extremities; and this appears to be generally from half an inch to an inch in children, and from one inch to an inch and a half or two inches in the adult; but it may occasionally exceed this, and yet retain its full strength and proportionate size to the extremities of the tendon it serves to connect. The greatest length I have witnessed in the human subject is two and a quarter inches, and this was in the tendo Achillis of an adult, a year and a half after the operation (Mr. Curling's case, above described).

This connecting bond of union, or new tendon, as it may with propriety be called, is of a very tough consistence, and appears to the naked eye to be homogeneous rather than fibrous. A small portion of it cannot be split into fibres, but when teased out with needles on a piece of glass for microscopical examination, it spreads out like a portion of

serous membrane, rather than splits into fibres, though its fibrous character is readily distinguishable under the microscope, as above described. There is, however, one very marked peculiarity of new tendon, even at a late period, viz., that, after the gradual subsidence of the vascular injection and consequent ruddy tinge of the new tendon, it presents to the naked eye a grayish translucent appearance, a peculiarity which it has retained up to the latest period I have had the opportunity of examining it in the human subject, viz., three years after operation; and in the rabbit, one year after operation. This grayish translucent appearance at once distinguishes the new from the old tendon in any specimen of which a section may be made in the recent state; but as the translucency is destroyed by spirit, which immediately renders the new tendinous tissue opaque, this indication is lost after the specimen has been put into spirit. Pure glycerine, or a solution of half glycerine and half water, preserves the appearance of translucency in the new tendon for a short time—a few weeks; but gradually this solution renders the old tendon translucent, and thus the contrast between the new and the old tendon is also lost. I am not aware of any fluid in which the appearance can be retained.

I would here observe that no section, in the recent state, had been made of the human tendons presented to the Royal College of Surgeons by Mr. Tamplin, upon the external appearances of which the theory of *a linear cicatrix*, supposed to result from the contraction and complete absorption of the new connective tissue, has been based. These tendons (the tendo Achillis, and tibial tendons of a child aged nineteen months, in whom these tendons had been divided nearly eighteen months previously[1]) are said not to have presented externally any appearance of having been divided, and minute portions, taken by Mr. Quekett from different parts, exhibited the microscopic appearances of well-formed tendon. Hence it appears to have been

[1] 'Path. Catalogue of the Museum of the Royal Coll. of Surgeons,' vol. ii,. Nos. 358, 359, and 360.

assumed by Mr. Tamplin that the new connective tissue had been completely absorbed, and that the divided extremities of the old tendon had again come into direct apposition, so that only *a linear cicatrix* remained. The fact that the new connective tissue, or new tendon, so closely resembles in structure the old tendon as to be scarcely distinguishable from it, had not at that time been demonstrated, and it is to be regretted that the recent section, the most important test, was not made. Sections of these tendons were made some months since by Mr. Quekett and myself, but from the effect of spirit in destroying the translucency of the new tendon (if such existed), it was impossible to say whether any new tendinous tissue existed or not. There did appear to me to be some indications of a portion of new tendon in the tendo Achillis, but the traces were obscure, and as the tendon of the opposite leg was not preserved, it is impossible to say whether any increased length of the tendon had been obtained.

4th. *Junction of the new with the old tendon.*—The divided extremities of the old tendon take no active part whatever in the reparative process during its earlier stages, and have but a slender connexion with the new material when first formed. A little later, certain changes are observed to occur in the divided extremities of the old tendon, and these commence earlier and proceed more rapidly in the upper than in the lower extremity of the tendon. These changes are—1st. The cut extremities of the tendon, with their square surfaces and sharp edges, become a little rounded, and their structure slightly softened. 2dly. They become slightly enlarged, and exhibit a disposition to split, and thin streaks of new material, of a grayish translucent appearance, are seen between the split fibres. This is the commencement of the junction between the old and the new tissues, and by the increase of new material between the split fibres of the old tendon, the extremity of the latter presents a slightly bulbous appearance. 3dly. At a later period this bulbous enlargement gradually diminishes, till the extremities of the old tendon again

assume their natural appearance, and the new and old tendon become of uniform diameter; but the appearance of a very fine dovetailing of the new material with the split fibres of the old tendon still remains, and in two cases above described was distinctly traceable in the recent section at intervals of a year and a half, and three years, after the division of the tendon.

This complete and firm junction of the new with the old tendon may be regarded as essentially the final stage of the reparative process; but proceeding simultaneously with it is a further perfecting of the structure of the new tendon, as to its density, the diminution of vascularity, general resemblance to the structure of healthy tendon, except in the translucency as above described, and in the external definition of form; but in all the specimens I have examined at late periods I have found a want of definition along the deep surface of the new tendon, arising from adhesions between this surface of the tendon and the deep fascia. In the human subject, the cellular tissue between the deep surface of the tendo Achillis and the deep fascia presents the appearance of having been infiltrated with lymph to a greater or less extent in different specimens, and its structure is proportionably increased in density and its fibrous septa thickened. It is no doubt this condition of deep-seated adhesions which limits the separation of the divided extremities of the tendon when subjected to a second operation, and hence arises the extreme importance of gaining sufficient length of new tendon after the first operation. When divided a second time, a tendon very rarely gives way with *a snap*, and the separation of its divided extremities is generally very slight. This I have found to be the case, even after an interval of ten years between the operations; but the adhesions would no doubt be much stronger when the first operation had been clumsily performed, and followed by an unnecessary amount of inflammation.

Amongst the changes which the new tendon undergoes in its gradual improvement at a late period, I have observed the more or less perfect reproduction of the sheath of the

tendon, which I described as forming in the first stage the matrix in which the reparative material is effused.

This process of re-formation of the cellular sheath on the surface of the new tendon is accomplished more perfectly, and is more easily traceable, in rabbits than in the human subject. In my experiments on rabbits I found a separable layer of areolar tissue formed on the surface of the new tendon through its entire length, between the second and third month after division; and above and below it was continuous with the cellular sheath surrounding the extremities of the old tendon, but it was neither so delicate nor so loose-textured as the cellular sheath of the old tendon; it adhered more closely to, and could not be so easily separated from, the surface of the new tendon. In dissecting from above downwards, or from below upwards, in either case from the old to the new tendon, and raising the investing cellular sheath, the line of junction of the new with the old tendon could be readily determined by this difference in the separability of the sheath, when no other indication was apparent.

In the human subject the same process takes place, but is not so easily demonstrated, owing to the existence of, and connexions of the sheath with the cellular tissue and fat in which the tendon is imbedded, and which is entirely absent in the rabbit.

Circumstances which may interfere with the perfection of the Reparative Process, or entirely prevent it, so that non-union of the divided Tendon may result.

Having now described the reparative process, as it appears to take place under the ordinary circumstances of health in a divided tendo Achillis, I would observe, that the process appears to be precisely similar in all other tendons which possess a cellular sheath and are similarly situated with respect to the surrounding soft tissues, or

which possess a delicate membranous sheath, as the flexor tendons in front of the wrist-joint, and tendons in many other situations. These delicate thecal membranes appear to serve the purpose of connecting the divided extremities of the tendons after section, and, together with the loose-textured areolar tissue (imperfect cellular sheaths) in immediate contact with the surface of the tendons, no doubt materially assist in the reparative process.

But there are some circumstances under which the reparative process may be interfered with to a greater or less extent, so that union may take place through an imperfectly formed and attenuated uniting medium, or, it is said, that union of the divided extremities of the tendo Achillis may completely fail. I have never seen a case of complete non-union in the tendo Achillis, and should feel disposed to doubt its occurrence, in consequence of the uninterrupted connexion of the cut extremities, which continues to exist after division through the medium of the sheath, which is never completely divided, and is often but little injured in a subcutaneous division; and if it should be completely divided, as in an open wound, the sheath does not retract with the tendon, in consequence of its connexion with the surrounding tissues, as above described. In the posterior tibial tendon, however, complete non-union does occur, from the absence of a connecting cellular sheath, and is probably of more frequent occurrence than may be supposed: but of this I shall presently speak.

The circumstances under which the reparative process in the tendo Achillis may be interfered with, and the union remain imperfect, are—

1st. Some constitutional defect in the reparative powers of the patient, or depressed vital power in the limb from paralysis.

2d. Injudicious after-treatment from—(*a*), not sustaining the temperature of the limb, especially in paralytic cases during very cold weather; (*b*), too early and too rapid mechanical extension, restoring the form of the foot before there is any evidence of the reparative material being

thrown out, or of the commencement of the reparative process; (*c*), the mechanical treatment being altogether ignored, and the patient allowed to walk within two or three days of the operation, the surgeon relying upon the unassisted powers of nature in walking to restore the form and functions of the foot;[1] (*d*), discontinuing the mechanical treatment too early, and bringing the foot too quickly into use, by which the uniting medium may be elongated and weakened.

From any of these circumstances imperfect union of the divided tendo Achillis may take place, the uniting medium being imperfectly formed and attenuated, in some cases consisting of little more than the sheath of the tendon somewhat thickened. Of the very serious results arising from this failure of the reparative process, not only in interfering with the restoration of the functions of the muscles, but in the production of talipes calcaneus, essentially an incurable deformity, it is not my intention to speak. The practical deduction, however, seems to be, that the rate at which the separation of the divided extremities of the tendo Achillis is to be produced by mechanical extension in the cure of deformities must be regulated by the activity of the reparative process. Rapid extension would not, I believe, endanger the perfect union of the tendo Achillis in well-nourished infants and children, in cases in which there is no paralysis of the limb operated upon. But in non-congenital cases of deformity at all ages, when any paralysis exists, as it certainly does in a very large proportion of such cases, the extension must always be slow and proportionate to the rapidity of the reparative process, which is evidenced by the filling up of the gap between the divided and separated extremities of the old tendon, and can be felt by external examination and manipulation.

[1] See 'Lancet' of 17th March, 1855, where this plan of treatment is advocated, and several cases given, by Professor Syme, of Edinburgh, in illustration of its supposed merits. The patients were encouraged to walk on the third day after the operation. (See also observations on this subject by Mr. Adams, in 'Medical Times and Gazette,' April 28th, 1855.)

Complete non-union of divided Tendons.

When tendons, situated *in dense tubular sheaths*, are divided, the reparative process appears liable to be more seriously interfered with, and if the divided extremities of the tendon are much separated, union sometimes completely fails, and the extremities of the tendon become adherent to the internal surface of the dense tubular sheath. I found this to have occurred in the posterior tibial tendon in one case, which I dissected two years after the tendon had been divided, and the appearances are represented in plate x, vol. viii, of the 'Transactions of the Pathological Society,' London, 1857.

In another *post-mortem* examination, which I made eleven days after division of the posterior tibial tendon, No. 2 of the present series, there seemed to be very little prospect of union taking place; the small portions of new tendon connected with the divided extremities of the old tendon were half an inch apart, without any connecting tissue between them. I have also seen good reason to believe in the failure of union in this tendon in several instances during life, in which, after its division, the feet have become flat and everted, and exhibit a degree of weakness and peculiarity of form which I believe to result from this cause.

With respect to the cause and the condition which favour the non-union of the posterior tibial tendon, I would observe, if the posterior tibial tendon be examined carefully as it passes through the dense tubular sheath behind the inner malleolus, a small quantity of very loose-textured and delicate areolar tissue, in the meshes of which small particles of fat are entangled, will be seen on its surface, and the same appearance will be noticed in other tendons in their passage through dense tubular sheaths; sometimes there is also an imperfect layer of very delicate thecal membrane.

Now, it will be obvious, from this anatomical examination alone, that if the posterior tibial tendon, or any tendon

similarly placed, be divided in a dense tubular sheath, and if the divided extremities of the tendon should separate, as I have shown they generally do, to the distance of half an inch, and sometimes to nearly an inch, there would be no tissue capable of holding in connexion these separated extremities. The small quantity of areolar tissue above described is not disposed as a regular cellular sheath, as there is no necessity for such a provision, but if the separation of the divided extremities of the tendon should not exceed a quarter of an inch, I think the areolar tissue described would be sufficient to connect them, and play the part of the cellular sheath of the tendo Achillis in forming the matrix in which the new reparative material may be effused: if, moreover, in a sudden and wide separation of the divided extremities this areolar tissue should be torn across, so that the ends of the tendon cease to have even this slender connexion, it would appear, from my examination in the case, No. 2 of this series, that the portions of this areolar tissue which remain connected with each end of the tendon may still serve as the matrix for the development of a small portion of new tendon connected with each extremity of the old tendon, but their junction may not take place.

In the case adverted to, the ends of the old tendon were separated to the extent of seven eighths of an inch, and connected with each extremity was a small portion of newly formed tendon, about a quarter of an inch in length. As there was no direct continuity between these two portions of new tendon, which were nearly half an inch apart, it seemed that there would have been no prospect of their ever coming into contact, and the only result of which I could see a probability was that of adhesion to the sheath, as in the case above adverted to, and described in the 'Pathological Society's Transactions,' vol. viii, plate x. Now, the question which arose in my mind with respect to the appearances presented in case No. 2 was, how were these portions of new tendon formed in connexion with the divided and completely separated extremities of the old

tendon? To suppose that the new material had been effused from the divided extremities of the old tendon would be in opposition to all that I have seen in my experiments on rabbits and in the human subject, and opposed to the opinion I have formed as to the part played in the reparative process by the extremities of the old tendon. The only explanation I could see of this abortive attempt at the formation of a connecting bond of new tendon was, that the small quantity of areolar tissue connected with each extremity of the divided tendon formed the matrix (as by its structure and vascularity it is undoubtedly capable of doing) in which the new material had been effused and developed, and it appeared to me that traces of this areolar tissue, with its included particles of fat, might be detected as now forming a component part of the small portions of connective tissue described.

Even when the posterior tibial tendon is divided a little above its dense tubular sheath, as was the case in the specimen just described, there seems to be a danger of non-union, when the separation is wide, in consequence of the lower extremity being drawn into the tubular portion of the sheath. Hence, in a case of varus, in which the posterior tibial tendon has been divided, it is clearly desirable not to straighten the foot quickly, where this is practicable; and in my opinion, at least a fortnight or three weeks should be allowed to elapse, in order to ensure union before the foot is brought into a straight line with the tibia.

These circumstances, then, interfere with the perfection of the reparative process of divided tendons, as required for the cure of deformities; and when we consider that the reparative effort required in tendons divided for the cure of deformities is not simply a joining of cut extremities, such as would be sufficient to unite a ruptured tendon (a process which might be compared with the union of bone), but consists in a perfect regeneration of tendinous tissue between the cut and purposely separated extremities of the old tendon, and that this newly developed portion of tendon is to form a permanent connecting bond of union, equal in

bulk and strength to the original tendon which it serves to connect, and from half an inch to two inches in length, our surprise will be, not that there are so many conditions likely to interfere with the perfection of this reparative process, but that under any conditions the process of regeneration of tendon is as perfect as I have shown it to be, when the required conditions exist, and are preserved by appropriate treatment.

The conclusions which I would deduce from the observations I have had the opportunity of making on the reparative process of tendons divided subcutaneously for the cure of deformities in the human subject, and above detailed, are as follows:

1st. That tendon is one of the few structures of the body capable of reproduction or regeneration, and that the newly formed tissue acquires, within a few months of its formation, the structural characters of the old tendon so perfectly as to be, under the microscope, with difficulty distinguishable from it; but it does not acquire (at least it has not up to three years, the latest period to which these observations extend) through its substance the uniformly opaque, pearly lustre of old tendon; in the mass it retains a grayish, translucent appearance, streaked only with opaque fibres at a late period, so that the recent section affords an easy method of distinguishing the new from the old tendon.

2d. That when a tendon has been divided subcutaneously, and its cut extremities are separated and held apart during the early stage of the reparative process, *i.e.* the first two or three weeks (as by mechanical extension employed with variable rapidity in different cases, according to certain rules in the cure of deformities) new tendon is formed, of variable length, according to the extent of the separation, for the purpose of reuniting the divided extremities of the old tendon. The greatest length of perfectly formed new tendon thus obtained, and equal in bulk and thickness to the tendon it served to unite, which I have

seen in the human being, is two inches and a quarter, and this was in the tendo Achillis of a girl, aged nine years, a year and a half after the tendon had been divided by Mr. Curling (Case 11 of the present series). It is probable that when the tendo Achillis has been divided in children for the cure of deformities, the length of new tendon is generally from half an inch to an inch, and in adults from one to two inches.

3d. That the process, by which new tendon is formed for the purpose of reuniting the separated extremities of a tendon divided by subcutaneous section, is essentially similar in animals and in man. That the perfection of the process is in direct proportion to the absence of extravasated blood and inflammatory exudation; and that the sheath of the tendon, when consisting of loose-textured areolar tissue, as in the tendo Achillis, and other tendons surrounded by soft tissues, is of importance—1st, in preserving a connexion between the divided extremities of the tendon; 2dly, in furnishing the matrix in which the nucleated blastematous, or proper reparative, material is effused; and 3dly, in giving definition and form to the newly developed tendinous tissue.

4th. That the perfection of the reparative process, especially in non-congenital cases of deformity, in which more or less paralysis frequently exists, may be interfered with by injudicious after-treatment, so that an elongated and attenuated uniting medium may be formed. And that when tendons situated in dense fibrous sheaths of a tubular form are divided, there is great danger of complete non-union, the divided extremities of the tendon becoming adherent to the inner surface of the sheath, without any direct connexion with each other. This I have shown to occur in the posterior tibial tendon, when divided immediately behind the inner malleolus.

The practical rule, therefore, is never to divide a tendon as it passes through the denser portions of its sheath, and when the operation is performed near to such portions of the sheath the extension must be conducted very slowly.

5th. That there is no reason for believing that, in the treatment of deformities by tenotomy, direct approximation and union of the divided extremities of the tendon must be first obtained, and that the required elongation is afterwards to be procured by gradual mechanical extension of the new connecting medium, as we should stretch a piece of india rubber; but, on the contrary, all my observations lead me to consider that the required length of new tendon should be obtained during the time occupied in its formation, *i. e.* from about two to three weeks under the ordinary conditions of health, but in paralytic cases this period may be doubled. Therefore, the object of gradual mechanical extension during this time, is to regulate the length of new tendon, by overcoming ligamentous resistance in some cases, especially in those of long standing and of congenital origin, in which it is difficult to separate sufficiently the divided extremities of the tendon; and by preventing excessive separation of the extremities of the tendon in other cases, especially in those of non-congenital origin, in many of which the ligaments offer no resistance, and the reparative power is very feeble from partial or complete paralysis, and also when tendons situated in or near dense tubular sheaths, as the posterior tibial, are divided. Hence the mechanical extension must always be proportionate to the activity of the reparative process, and in the tendo Achillis, and other superficial tendons, this can be judged of by external examination and manipulation.

6th. That the new tendon remains during life as a permanent tissue, and as an integral portion of the tendon, the divided extremities of which it has been formed to reunite.

I see no reason for believing that the newly formed tendinous structure has any disposition to undergo a process of gradual contraction, such as we see taking place in the cicatrices of the skin after burns, to which it has been compared; and that ultimately it becomes absorbed, the muscular structure at the same time becoming elongated by the force of the contraction of the cicatrix, so as to allow of the

re-approximation of the ends of the divided tendon, and the formation of a "*linear cicatrix.*"

Difficult as it may be to conceive the occurrence of this process in infants, in whom the muscular structures are all essentially healthy, it appears to me to be impossible to conceive its occurrence in cases of paralytic deformity, in which the muscles of the calf are found to be in a most advanced stage of fatty degeneration, so that under the microscope scarcely a trace of muscular tissue can be found, this being replaced by fibrous and fatty tissue.

The explanation given of the mode in which deformities are supposed to be cured upon this *linear cicatrix* theory, viz., by "an altered sphere of muscular action," does not appear to be at all intelligible. Yet this is the theory at present in vogue, having been originally propounded, and being still maintained, by my colleague, Mr. Tamplin,[1] as a modification of the views of Stromeyer, and supported by my colleagues, Messrs. Brodhurst[2] and Coote.[3]

Upon this *linear cicatrix* theory I am utterly unable to understand the use of tenotomy in the cure of deformities; whereas, in the facts demonstrated in the present communication, we see sufficient evidence that, in the cure of deformities, muscles are elongated by the increased length of their tendons, obtained by means of subcutaneous division, and the development of new tendon formed for the purpose of reuniting the divided extremities of the old tendon.

7th. That the effect of this permanent elongation of the tendons of contracted muscles, or, more properly speaking, muscles shortened by adapted growth, or adapted atrophy, is not only to correct deformities mechanically, by allowing certain bones to be brought into their normal anatomical relations; but that, having obtained this end, its higher physiological object is to allow of motion being gained or, as it may happen to be, regained, in joints which were previously rendered fixed and motionless by the contraction

[1] 'On Deformities,' p. 5.

[2] 'On Club-foot,' p. 103.

[3] 'Med. Times and Gazette,' Jan. 9th, 1858.

and structural shortening of the muscles. The mechanical conditions necessary for motion are thus obtained, and if the muscles themselves are in a healthy condition, voluntary motion is also obtained, and is followed by an increased development of the muscular structure, so that the bellies of the muscles operated upon progressively enlarge, and in a case of congenital talipes varus the gastrocnemius and soleus muscles, thus brought into use, attain in a few years a very fair proportionate size, instead of undergoing a process of progressive atrophy, as is seen in cases of club-foot which have remained unoperated upon till the period of youth, or adult life. The effect of the operation in increasing the size of the calf is very conspicuous, when a case, which has been successfully operated upon at an early age, is compared with a case which has not been operated upon, or subjected to other treatment; and it is thus proved that, when the possibility of muscular improvement exists, the muscular strength of the limb is very materially increased, instead of being diminished, by the operation of tenotomy. The extent to which muscular power is restored, with its attendant advantages, must of course depend upon, and be proportionate to, the healthy condition of the muscles, and *vice versâ;* so that, in cases of complete atrophy and degeneration of the muscular structure, as in old paralytic cases, the results of the permanent elongation of the tendons is purely mechanical.

8th. That when recontraction of the foot takes place, and the deformity returns at a distant period after tenotomy, this does not depend upon absorption of the new material, or new tendinous tissue formed previously to unite the divided extremities of the old tendon, but upon structural alterations taking place in the muscular tissue, either of an active character, as in spasmodic cases, or of a passive character, as in paralytic cases, and cases produced by position, &c., in which the muscles, by a process of *adapted atrophy*, as described by Mr. Paget, simply adapt themselves to the altered mechanical relations of the parts with which they are connected.

In three cases of relapsed deformity of the foot examined by me, Nos. 10, 11, and 12, of the present series, the new tendinous tissue formed after the previous operations remained, preserving its translucency and other characters, by which it could be easily distinguished from the old tendon. This fact must be regarded as additional, and I may say conclusive, evidence against the soundness of the *linear cicatrix* theory, and the supposed temporary nature of the new material above adverted to.

9th. That the ultimate perfection of the reparative process by the regeneration of tendinous structure, and the elongation of a shortened muscle by the insertion of a portion of new tendon into its length, equal in strength to the old tendon, and closely resembling it in its microscopic characters, is marred only by the adhesion of the deep surface of the new tendon to a greater or less extent with the neighbouring fibro-cellular tissue. These adhesions may limit the free play of the tendon, but will not interfere with sufficient motion being obtained. In cases of relapsed deformity, however, these adhesions will prevent sufficient separation of the divided extremities of the tendon being obtained. Therefore, if much separation be required, a second operation is generally unsatisfactory in its result, and beyond a second operation very little advantage can ever be obtained from operative treatment. Hence the necessity of the closest attention to the treatment after the first operation, and the explanation of the complete failure after repeated operations for the cure of deformities. The adhesions after several operations were so numerous and so strong along the tendo Achillis in Case 12, that no further benefit could have been obtained by tenotomy.

OBSERVATIONS

ON THE

MEDICAL ADMINISTRATION

OF

OZONIZED OILS.

BY

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Received May 23d.—Read June 28th, 1859.

Ozone, since its discovery by Schönbein, fifteen years ago, has proved an object of increasing interest, both to the chemist and to the physiologist. Air unduly charged with it is capable of acting strongly on the mucous membrane, rendering respiration difficult, and in some animals even producing fatal effects. The natural presence of ozone in moderate proportion in the atmosphere appears, however, to prove highly beneficial to the human system, and it seems therefore reasonable to inquire whether, if this agent could be medicinally administered in a definite and graduated manner, some remedial effects might not be obtained. Mr. Dugald Campbell, the analytical chemist to the Consumption Hospital, suggested to me that its administration might possibly be effected through the medium of ozonized oils, which he obligingly prepared for me by saturating cer-

tain oils with oxygen and then exposing them for a considerable time to the direct rays of the sun. It may be interesting to remark that one indication of the satisfactory progress of the operation is the appearance of a peculiar, acid, watery-looking fluid at the bottom of the vessel; it may also be mentioned that the fact of the oils being charged with ozone can be rendered obvious by the usual tests, viz., by the bleaching properties which they possess, and the action which they exert upon preparations of iodide of potassium and starch.

During the last few years I have from time to time administered oils thus prepared, and I now offer to the Society a short and simple statement of the results observed in all the patients to whom they were given, of whose cases sufficient records have been preserved.

John H—, a labourer, æt. 33, having phthisis in the first stage, as indicated by dull percussion, prolonged expiratory murmur, wavy inspiration, and increased vocal resonance on the right side, was admitted on the 8th of October, 1855; he took salines for two days, afterwards two drachms of ozonized sunflower oil twice a day. Under the use of the medicine his pulse was reduced in a few days from 104 to 92. He left the hospital on the 22d of October, in improved health, but without any increase in weight.

Frederick C—, æt. 18, five feet eight inches in height, weight seven stone twelve and a quarter pounds, was admitted on the 4th of October, affected with phthisis in the first stage. He took salines for six days. On the 10th, the pulse being then 118, he was ordered to take two drachms of ozonized sunflower oil three times a day; on the 20th the pulse had fallen to 102, on the 24th to 86, and his weight had increased to seven stone thirteen and a half pounds. The pulse continued low under the use of ozonized cocoa oil, but on the 31st of October, the supply of ozonized oil being exhausted, simple oil was substi-

tuted, and on the 3d of November the pulse had risen to 108.

Mary T—, admitted October the 2d, with a pulse at 108, commenced the use of ozonized sunflower oil on the 10th. This patient's pulse, on the 29th, was reduced to 100, but her weight remained unchanged.

Joseph S—, æt. 20, admitted the 17th of October, 1856, having, among other symptoms, crackle (dry crepitation) at the apex of the left lung, and venous hum in the right jugular, was treated with simple saline medicines till the 21st of October, when he was directed to take two drachms of coarse cod-liver oil, ozonized, twice a day. This patient's height was five feet eleven and a half inches, his weight ten stone, pulse 108. On the 25th the pulse was 88, on the 28th, 90, and on the 3d of November it had risen to 102; the dose of ozonized oil was then increased to half an ounce; the cough and expectoration continued unchanged, but other symptoms improved, and under the augmented dose of ozonized oil the pulse again slightly fell.

William B—, æt. 8, with a vomica at the upper part of the right lung and some tubercular deposition in the left, took ozonized coarse cod-liver oil twice a day, from the 30th of October to the 3d of November, the pulse falling from 120 to 100 during those three days; the medicine was then administered three times a day, and in less than a week the pulse was reduced to 80.

Samuel H—, æt. 28, having vomica on the left and softened tubercle on the right side, took coarse ozonized cod-liver oil from the 29th of October to the 8th of November, but the pulse was not influenced, remaining about 90 throughout.

Sarah W—, æt. 23, admitted October the 22d, took ozonized coarse cod-liver oil from the 25th of October to

the 2d of December; her pulse continued about 108 throughout, but she felt better and increased in weight from seven stone eight pounds to seven stone thirteen and a half pounds. In this patient there was murmur in the jugular vein, a symptom indicative of a condition of the system probably favorable to the efficiency of cod-liver oil.

David J—, having tubercular deposit in both lungs, which had advanced to softening on the left side, was admitted September the 22d, 1857; his height was five feet seven inches, and his weight nine stone one and a half pounds. He took simple cod-liver oil till the 10th of December, his weight being then ten stone and his pulse 112; he was then ordered ozonized cod-liver oil, and his pulse, with one slight fluctuation, gradually fell to 76. On the 6th of January simple cod-liver oil was substituted, but the pulse being on the 13th of January 108, the ozonized oil was resumed. On the 15th, 17th, and 19th of January the pulse varied from 88 to 84; simple coco-oleine was then substituted, and the pulse rose in a few days to 92. On the 3d of February ozonized coco-oleine was administered, and on the 6th the pulse was 86.

Thomas R—, æt. 15, was admitted December the 2d, 1857, having tubercular deposit on the left side, as indicated mainly by dull percussion and harsh respiration. He commenced the use of ozonized cod-liver oil, in doses of two drachms twice a day, on the 8th of January, 1858, the pulse being at that time 95; on the 13th the pulse had risen to 104; the dose of ozonized oil was then increased to half an ounce, and in a few days the pulse had fallen to 86.

Thomas W—, æt. 17, in an early stage of phthisis, was admitted on the 14th of December, 1857. His height was five feet five inches, weight nine stone four pounds, respirations 26. He took two drachms of simple cod-liver oil

twice a day till the 6th of January, when, the pulse keeping up steadily about 90, the same dose of ozonized cod-liver oil was substituted; on the 3d of February the pulse had fallen to 68.

Charles T—, æt. 43, admitted on the 12th of January, 1858, had vomica on the right side and tubercular deposition on the left. He had suffered from cough and expectoration for two years, and presented other evidences of advanced consumption, being so much emaciated as to weigh only seven stone nine and a half pounds, although his height was five feet four inches. He was treated in succession with salines and mixture of tolu; on the 20th of January the pulse was 80; on the 23d, the pulse being at 86, three drachms of cod-liver oil were given twice a day; the pulse, with some fluctuations, became decidedly quicker, and on the 28th of January was 92. Three drachms of ozonized coco-oleine were then substituted; on the 1st of February the pulse was 88, and on the 3d, 80.

Jane J—, æt. 26, a very nervous patient, having arterial murmur and venous hum, but with slight indications of the presence of phthisis in the first stage, was admitted on the 9th of December, 1857. She was at first treated with ammonia, cascarilla, and prussic acid; but the pulse rising under this treatment, digitalis and aconite were administered, without, however, any permanent effect in lowering the circulation. On the 7th of January quassia and iron were given, and on the 13th ammoniated cascarilla mixture was resumed. On the 27th of January ozonized coco-oleine was given twice a day, in two-drachm doses. The pulse, which on the 29th of January was 104, on the 2d of February had fallen to 88, but on the 6th it had risen to 112. It is possible that an increased dose of the ozonized oil, if at this time administered, might have maintained a more decided effect on the pulse, but any conclusions derived

from the state of the circulation regarding the action of medicines on this patient are rendered very uncertain by her highly nervous temperament.

Susan F—, admitted March the 16th, 1858, took cod-liver oil for a week, without any obvious effect on the pulse; ozonized oil was then given, and the pulse remained for several weeks during its use about 100, but on the discontinuance of the ozone speedily rose to 110.

In another example, of which, however, I have not preserved full details, after the pulse had fallen considerably under the use of ozonized oil, I observed at one of my visits that the pulse had risen; but on desiring Mr. James Ford, my clinical assistant, who took an active interest in these investigations, to record it as an exceptional case, he replied: "There is an omission in the register; this patient has not had ozonized oil the last three days, for the supply is exhausted."

For facility of reference, the more obvious results are appended in a tabular form. The few instances in which the number of respirations in the minute is recorded are not sufficient to establish any conclusion, but they certainly do not indicate that the remedy had any particular influence in this respect.

During my absence from the hospital my friend Dr. Scott Alison administered ozonized oil to some of the patients. I am happy to be allowed to append his reply to my inquiry regarding the result of his experiments, and to find that his independent observations are corroborative of my own. Dr. Alison remarks: "Under my direction only four of the patients had had ozonized oil given in such a manner as to be likely to give any result. Unfortunately, all the patients were in the third stage of the disease, and no lasting benefit was experienced.

"In two cases a remarkable reduction in the rapidity of the pulse occurred under the use of the ozonized oil, the reduction amounting to about twenty beats, while this improvement could not be referred to any other cause. I attach some value to this observation, for I prescribed the oil totally divested of all prejudice in its favour, and I have always been reluctant, on imperfect grounds, to refer results to the operation of medicine. The oil was given in the doses employed by yourself. If ozonized oil can reduce the rapidity of the circulation, a feature of great prominence in phthisis, this remedy possesses a most valuable property, rendered still more valuable by its contributing at the same time to improve the general health."

In order fully to appreciate the evidence adduced in this paper, it is important to observe that in some of the patients the use of simple and of ozonized oil was more than once alternated, with corresponding variation of effect. In the case of David J— the alternation was made three times, and the result was, in each interchange of treatment, so direct and remarkable, as to make that particular example equivalent in force to three experiments. It is difficult to review the history now presented without being impressed with the conviction that the administration of ozonized oils has a remarkable tendency to reduce the frequency of the pulse.

Of the fourteen patients whose cases are detailed in this communication there are only two in whom no such effect was observed, and although in a few instances the effect may have seemed insignificant or transient, in the larger proportion it was very considerable, and must be attributed to the ozone rather than to the oil, since it was repeatedly manifested in patients who had taken cod-liver and other oils without any reduction, or even with an acceleration of pulse; and further, the effect on the pulse was nearly as distinct when the ozone was associated with the oil of the

cocoa-nut or of the sunflower,[1] as with that of the cod-liver. This circumstance is the more significant since the administration of sunflower oil without ozone has not appeared to me to manifest any important remedial power. The reduction of pulse in the cases described in this communication was usually observable in two or three days, and was often progressively maintained for a considerable period. A reduction of twenty beats was observed in certain cases to occur respectively in two, three, four, and six days; in other instances a reduction was noted of twenty-four pulsations in fourteen days, thirty-four in thirteen, thirty-six in twenty-two, forty in eleven. In one patient the pulse fell as low as 60, probably considerably below the natural standard, but in most of the favorable instances the reduction stopped when that standard was obtained. The apparent effect of the remedy is one which, prior to experiment, I should not have anticipated. It was not attended by any other obvious result, excepting that a general improvement of the patient's condition seemed to be associated with it.

I have used ozonized oil of turpentine in some cases of hæmoptysis with apparently prompt advantage, but have not sufficiently repeated the experiment to be entitled to express a positive opinion as regards the remedial superiority of ozonized over ordinary turpentine. Should more extended observation establish for ozonized oil the property which these records appear to indicate, it will prove a valuable addition to our remedial means;—*specially*, it may be, in consumption, which is a disease peculiarly characterised by hurried action, but not perhaps exclusively in this disorder, since there are other morbid conditions, in the treatment of which it is very important to lower the pulse without reducing power. It is true that additions to our list of medicines are not generally to be desired. If the

[1] For the careful preparation of the cocoa and sunflower oils, I am indebted to the scientific zeal and friendly aid of Mr. Fergusson Wilson.

extension of therapeutics is ever to become proportional to that of other departments of medical science, it will be by a more successful investigation of the modes of action of familiar remedies and a more precise discrimination of the conditions which should regulate their employment; and with the progress of real knowledge in this direction, the number of medicines in daily use will probably, as a consequence, be lessened. Nevertheless, the substance recommended in this communication has more than a common claim to consideration, since it is now generally regarded as an active form of oxygen, an element intimately connected with the economy of life.

Case.	Stage.	Date.	Medicine.	Pulse.		Respiration.	Weight, &c.
		1858		Sitting.	Standing.		
C. T.	Third on right side, first on left	Jan. 12	Salines.....	...	...	...	7st. 9½lbs. (5 ft. 4 in.)
		Jan. 18	Tolu mixture				
		Jan. 20	...	80	86	16	
		Jan. 23	Cod-liver oil, ʒiij, twice daily	86	96		
		Jan. 27	...	92	100	20	
				104	120	20	(After exercise.)
		Jan. 28	Ozon. coco-oleine, ʒiij	92	100		
		Feb. 1	...	88	100	20	
		Feb. 3	...	80	96		
		1857					
J. J.	First	Dec. 9	Ammonia, cascarilla, prussic acid Digitalis; aconite	...	...	...	8st. 11lb. (Venous hum, arterial murmur.)
		1858					
		Jan. 7	Quassia, iron				
		Jan. 13	Ammonia, &c.				
		Jan. 27	Ozon. coco-oleine, ʒij, twice a day	108	122		
		Jan. 29	...	104	120		
		Feb. 2	...	88	96		
		Feb. 6	...	112	128		

ON THE CONNEXION

BETWEEN THE

HEAT OF THE BODY

AND THE

EXCRETED AMOUNTS OF UREA, CHLORIDE OF SODIUM, AND URINARY WATER,

DURING A FIT OF AGUE.[1]

BY

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COMMUNICATED BY

RICHARD QUAIN, F.R.S.

Received June 6th—Read June 23th, 1859.

THE observations on the temperature of the body during ague fits are now numerous and accordant,[2] and may be considered sufficient to indicate the general condition of the animal heat during the several stages of ague.

[1] The following observations were made at the suggestion of Dr. Parkes, from whom, from time to time, I have received assistance. Dr. Parkes has also supplied the entire literature in the paper, and has kindly examined it with care.

[2] Zimmermann; Bärensprung, Müller's 'Archiv,' 1852, p. 217; Michael, 'Archiv für Phys. Heilk.,' 1856, p. 39; Wunderlich, 'Archiv für Phys. Heilk.,' 1858, p. 12.

The increase in the amount of urea and of chloride of sodium during the cold and hot stages has been affirmed by Traube and Jochmann,[1] Moos,[2] Redenbacher,[3] and Hammond.[4] These observations have been criticised, however, and certainly some of the cases were not examined with any great minuteness. In no case yet reported either has any comparison been drawn between the rise in the temperature and the amount of the urea and chloride of sodium.[5] It therefore seemed extremely desirable, on the occasion of a patient with ague being admitted into University College Hospital, under the care of Dr. Parkes, to examine the subject, and to see, first of all, whether the increase of urea and of chloride of sodium really occur in the cold and hot stages of ague, as affirmed by Traube, Redenbacher, and Hammond; and, secondly, whether any close connexion could be traced between the amount of urea and the abnormal temperature. My position as one of the resident officers in the hospital at the time gave me the opportunity of carrying out the inquiry with all the minuteness necessary for accuracy.

The general results may be thus stated—that not only was the increase of urea and of chloride of sodium constant during the cold and hot stages of ague, but that their amount was in very close relation to the temperature. The first case recorded in this paper will, I believe, give little short of mathematical proof of the connexion between these two phenomena, viz., the increase of heat of body and of the excretion of urea and chloride of sodium.

The second case communicated is one in which the phenomena were less carefully observed, but it is valuable as affording another instance of the increase of urea and chloride of sodium during the fit.

[1] 'Deutsche Klinik,' No. 46, Nov., 1855.

[2] Henle's 'Zeitschrift für rat. Med.,' Band vii, p. 291.

[3] Henle's 'Zeitschrift,' Band ii (Dritte Reihe), p. 384.

[4] 'American Journal of Med. Science,' April, 1858.

[5] Some other observations have been made on the excretions of twenty-four hours, but these are of little value as febrile and non-febrile bones are put together.

In addition, a case of hectic fever occurring in phthisis has been narrated, for in this case the phenomena were found to be identical with those presented by malarial ague. The result was the same in both cases, although the causes were so different.

Case 1. *Quotidian ague.*—The patient, a man, was first attacked by tertian in August last, whilst working at Maldon, in Essex. The ague continued on him for three weeks, by which time he was apparently cured; he remained working at the same place till the December following. At that time, moving up to London, he broke his leg, and was carried to Charing Cross Hospital. Upon being discharged from that institution, cured, he caught cold, and an attack of ague immediately followed, and has continued more or less since, being immediately brought on by exposure to cold. This long continuance produced the usual effects of prolonged ague; and in this state he was admitted into the hospital, the disease having changed a day or two previously from the tertian to the quotidian type.

He suffers from slight aortic obstructive disease. His pulse is continually about 120 per minute,[1] exceedingly irregular in both force and rhythm. His arteries are very tortuous and visible. He never suffered from rheumatism, and has no arcus senilis. Except the above heart-disease, he suffers from no organic lesion.

He is fifty-nine years of age, but looks much older. His weight is 144 pounds; his height five feet nine and three quarter inches. His vital capacity (Hutchinson's spirometer) is 140 cubic inches.

The examination was conducted in the following manner. The patient was put to bed, and his urine collected through the night. At 5 a.m. he had breakfast, consisting of two eggs, bread and butter, and tea, the latter being measured. At 6 a.m. he was made to pass his urine, and all passed at

[1] No tables are therefore given of the pulse.

the time was mixed with that passed during the night. The thermometer (a good one, by Negretti and Zambra, and divided into fifths of a Fahrenheit degree) was next placed in his axilla, and kept there during the remainder of the day, the temperature being noted down every quarter of an hour. He was also made to pass his urine hourly, or upon the commencement of another stage, before the completion of the hour, and the next hour was dated from that time; by this means we not only obtained the urine of every hour, but also of each stage, separately. The patient was allowed no food until the completion of the fit, when he had a good meat dinner and six ounces of wine. Lemonade he was permitted to drink *ad libitum*, but the quantity was always measured and noted, with the time at which it was taken. He was weighed immediately after his breakfast, and again at the termination of the fit, and during this time no motions were allowed to be passed. The patient was purposely kept without medicine. In determining the amount of urea and chloride of sodium, Liebig's volumetric method with nitrate of mercury was used.

It was not possible to determine the other urinary constituents in this case. The chloride of sodium was not got rid of before testing for urea, but the usual correction was made.

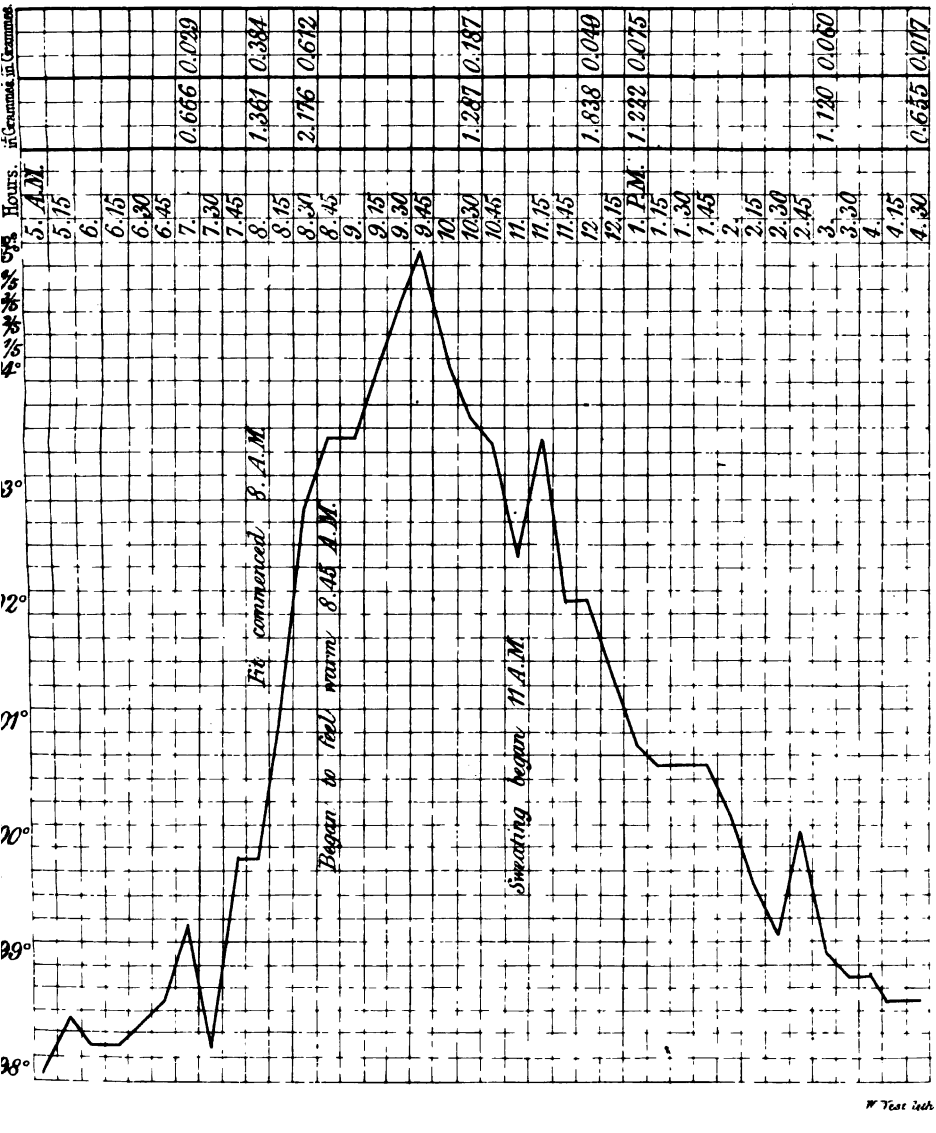
The following charts show the variations in the temperature in fifths of a degree, as taken every quarter of an hour. Above the table are two columns, one showing the amount of urea, the other the amount of chloride of sodium, poured out. The quantity per hour is stated in each case, and put down in the column denoting the time at which it was passed. It indicates, of course, the quantity formed during the previous hour. The amounts of urea and chloride of sodium are given always in French grammes, and the water in cubic centimetres. The commencement of each stage, as judged of in the usual way, by the sensations of the patient, is also noted in the column proper to the time at which it began.

For the purpose of greater clearness and of giving fuller

APRIL 6TH

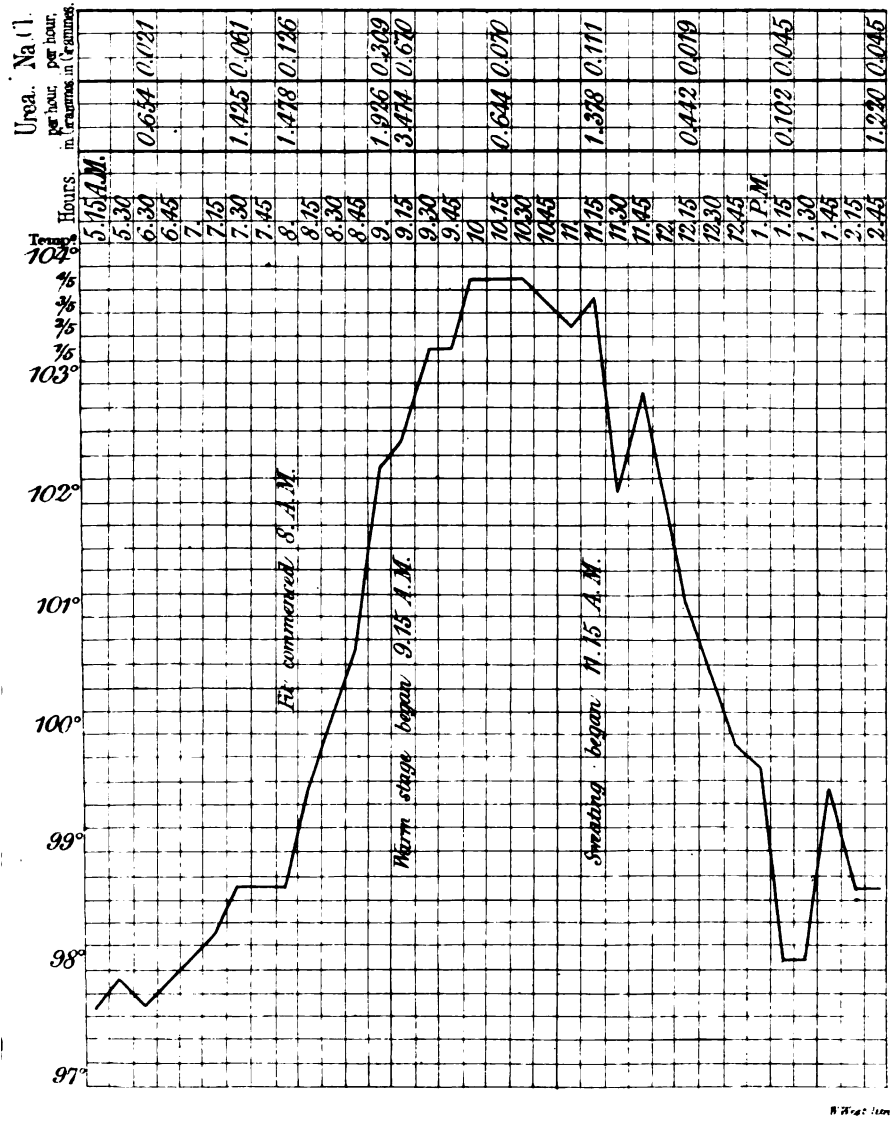
7. A.M.
7.15
7.30
7.45
8.15
8.30
8.45
9.
9.15
9.30
9.45
10.
10.15
10.30
10.45
11.
11.15
11.30
11.45
12.
12.15
12.30
12.45
1. P.M.
1.15
1.30
1.45
2.
2.15
2.30
2.45
3
3.15
3.30
3.45
4.
4.15
4.30
5.30
1.635 0.135
1.984 0.276
2.088 0.230
1.820 0.130
1.298 0.106
1.620 0.162
0.993 0.064
0.848 0.063
2.037 0.145
Ht commenced 8. A.M.
Hot stage commenced 8.30 A.M.
Sweating commenced 11.15 A.M.
7. P.M.
8.
9.
10.
11.
12.
1. A.M.
2.
3.
4.
5.
6.
7.
98 4/5
98 3/5
98 2/5
98 1/5
98

APRIL 7TH



W Test lith

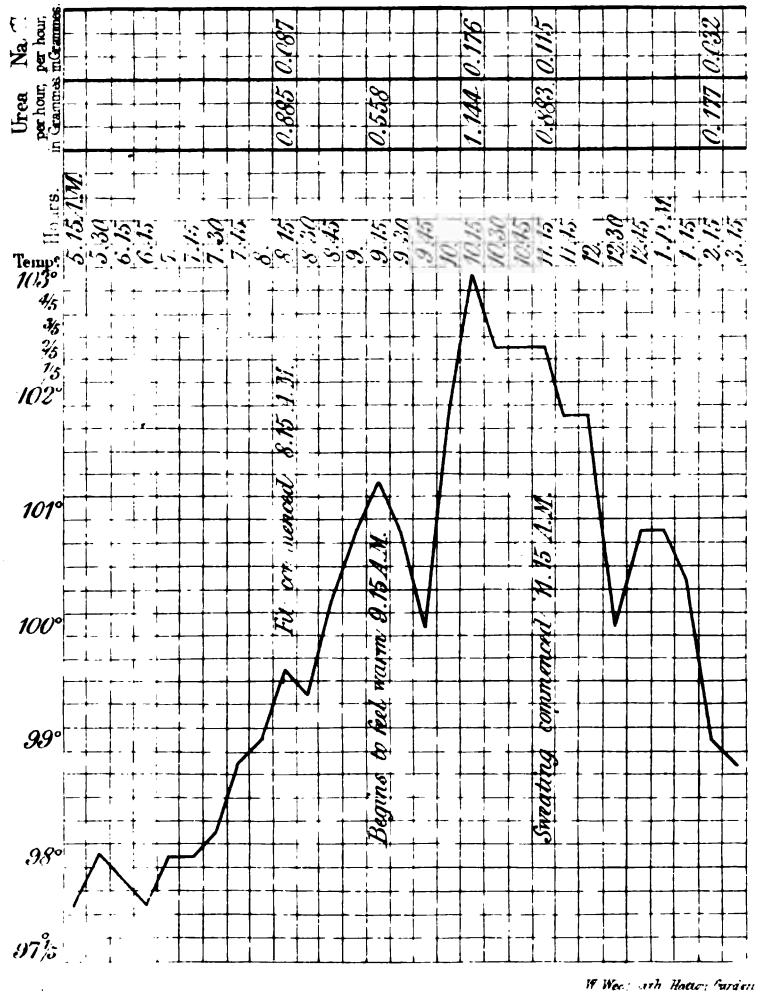
APRIL 8TH


Urea. Na Cl.
Urea, per hour, in grammes.
Na Cl, per hour, in grammes.
Hours
Temps
5.15 A.M.
5.30
6.45
7.
7.15
7.30
7.45
8.
8.15
8.30
8.45
9.
9.15
9.30
9.45
10.
10.15
10.30
10.45
11.
11.15
11.30
11.45
12.
12.15
12.30
12.45
1. P.M.
1.15
1.30
1.45
2.15
2.45
0.634 0.021
1.135 0.061
1.418 0.126
1.926 0.309
3.474 0.670
0.644 0.070
1.338 0.111
0.442 0.079
0.102 0.045
1.240 0.045
104°
⅘
⅗
⅖
⅕
103°
102°
101°
100°
99°
98°
97°
Ft commenced 8 A.M.
Warm stage began 9.15 A.M.
Sweating began 11.15 A.M.

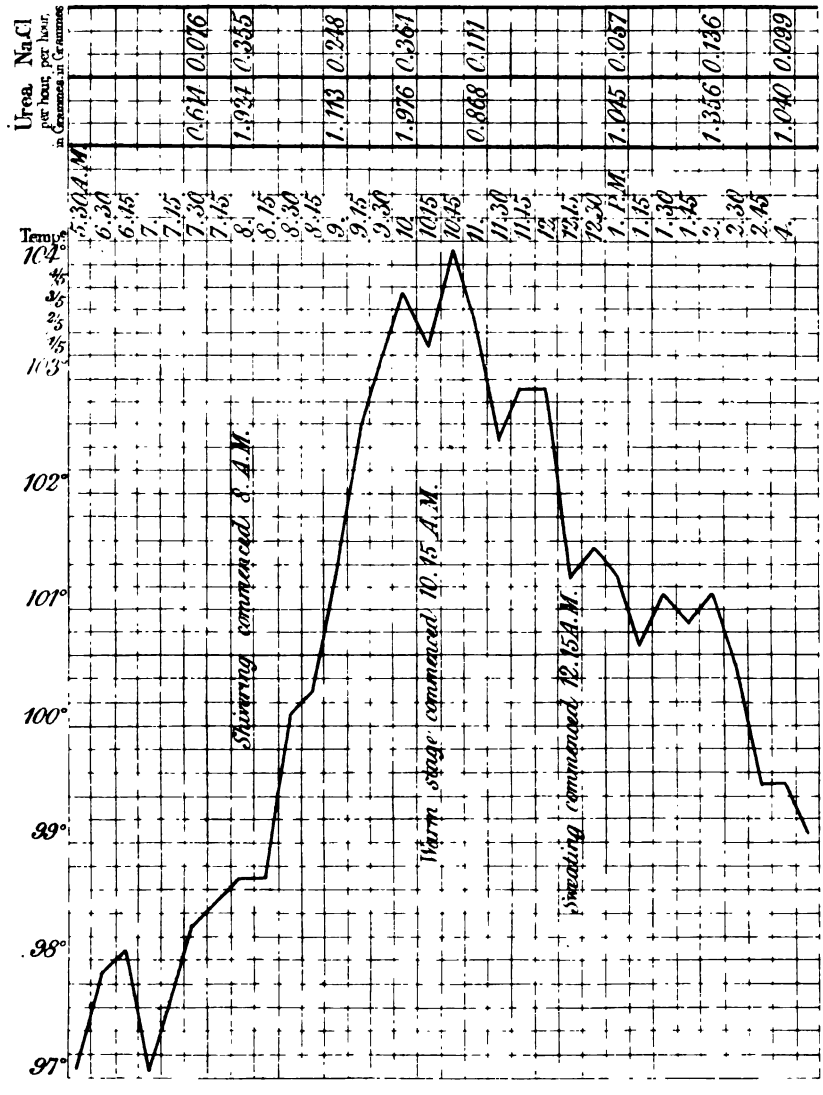
APRIL 9TH

Urea. Na Cl.
per hour, in Grammes.
Hours.
Temp.
5.30 A.M.
6.30
6.45
7
7.15
7.30
7.45
8.
8.15
8.30
9.
9.15
9.30
9.45
10.
10.15
10.30
10.45
11.
11.30
11.45
12.
12.15
12.30
12.45
1. P.M.
1.30
1.45
2.
2.15
2.30
2.45
3.
3.15
3.30
0.927 0.163
1.351 0.155
1.089 0.231
2.132 0.561
1.268 0.140
1.032 0.092
1.096 0.063
104°
103°
102°
101°
100°
99°
98°
97½°
Fit commenced 7.45 A.M.
Hot stage began 9. A.M.
Sweating began 11. A.M.

APRIL 10TH


Urea Na. Cl
per hour, per hour,
in Grammes. in Grammes
Hours.
Temp°
105°
4/5
3/5
2/5
1/5
102°
101°
100°
99°
98°
97⅘
5.15 A.M.
5.30
6.15
6.45
7.
7.15
7.30
7.45
8.15
8.30
8.45
9.
9.15
9.30
9.45
10.
10.15
10.30
10.45
11.15
11.45
12.
12.30
12.45
1.
1.15 P.M.
1.15
2.15
3.15
0.885 0.087
0.558
1.444 0.176
0.883 0.115
0.177 0.032
Fit commenced 8.15 A.M.
Begins to feel warm 9.15 A.M.
Sweating commenced 11.15 A.M.
W Wes[illegible]orth Hooker Carrier

APRIL 11TH



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details, tables are added, recapitulating the amount of the urea, chloride of sodium, and water, separately. In the first column of each table the stages are written down; in the next the hour at which the urine was passed; then follows the total amount of urea contained in the urine passed during the specified time; the next column shows the amount reduced to hours; and, lastly, the average amount of each stage per hour is given.

TABLE.

APRIL 6th.—UREA.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
Cold	7·45 to 8·30 a.m.	1·356	1·695	1·695
Hot	8·30 to 9·30 a.m.	1·984	1·984	
	9·30 to 10·30 a.m.	2·088	2·088	1·964
	10·30 to 11 a.m.	0·910	1·820	
Sweating	11 to 12 a.m.	1·289	1·289	
	12 to 1 p.m.	1·620	1·620	
	1 to 2 p.m.	0·993	0·993	1·363
	2 to 3 p.m.	0·848	0·848	
	3 to 4 p.m.	2·057	2·057	
		CHLORIDE OF SODIUM.		
Cold	7·45 to 8·30 a.m.	0·108	0·135	0·135
Hot	8·30 to 9·30 a.m.	0·216	0·216	
	9·30 to 10·30 a.m.	0·270	0·270	0·220
	10·30 to 11 a.m.	0·065	0·130	
Sweating	11 to 12 a.m.	0·106	0·106	
	12 to 1 p.m.	0·162	0·162	
	1 to 2 p.m.	0·064	0·064	0·108
	2 to 3 p.m.	0·063	0·063	
	3 to 4 p.m.	0·145	0·145	
		WATER.		
Cold	7·45 to 8·30 a.m.	75 c.c.		
	8·30 to 9·30 a.m.	80 c.c.		
Hot	9·30 to 10·30 a.m.	90 c.c.		
	10·30 to 11 a.m.	82 c.c.		
Sweating	11 to 12 a.m.	59 c.c.		
	12 to 1 p.m.	90 c.c.		
	1 to 2 p.m.	54 c.c.		
	2 to 3 p.m.	53 c.c.		
	3 to 4 p.m.	121 c.c.		

APRIL 7th.—UREA.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
During night...	3 p.m. to 5·30 a.m.	10·272	0 684	0·684
Period immediately before shivering	5·30 to 7 a.m.	0·999	0·666	0·666
	7 to 8 a.m.	1·361	1·361	1·361
Cold	8 to 8·30 a.m.	1·088	2·176	2·176
Hot	8·30 to 10·30 a.m.	2·575	1·287	1·287
Sweating	11 to 12 a.m.	0·838	0·838	
	12 to 1 p.m.	1·222	1·222	0·935
	1 to 3 p.m.	2·240	1·120	
	3 to 5 p.m.	1·311	0·655	

CHLORIDE OF SODIUM.

During night...	3 p.m. to 5.30 a.m.	0·642	0·029	0·029
Period before shivering	5·30 to 7 a.m.	0·440	0·29	0·29
	7 to 8 a.m.	0·384	0·384	0·384
Cold	8 to 8·30 a.m.	0·306	0·612	0·612
Hot	8·30 to 10·30 a.m.	0·375	0·187	0·187
	11 to 12 a.m.	0·049	0·049	
	12 to 1 p.m.	0·075	0·075	0·064
	1 to 3 p.m.	0·120	0·060	
	3 to 5 p.m.	0·034	0·017	

QUANTITY OF URINE PASSED.

	3 p.m. to 5·30 a.m.	36 c.c.		
Beforeshivering	5·30 to 7 a.m.	25 c.c.		
	7 to 8 a.m.	80 c.c.		
Cold	8 to 8·30 a.m.	136 c.c.		
Hot	8·30 to 10·30 a.m.	62 c.c.		
	11 to 12 a.m.	33 c.c.		
	12 to 1 p.m.	50 c.c.		
	1 to 3 p.m.	50 c.c.		
	3 to 5 p.m.	28 c.c.		

APRIL 8th.—UREA.

Period immediately before shivering	5 p.m. to 6·30 a.m.	8·832	0·654	0·654
	6·30 to 7·30 a.m.	1·425	1·425	1·452
	7·30 to 8 a.m.	0·738	1·478	
Cold	8 to 9 a.m.	1·926	1·926	2·700
	9 to 9·15 a.m.	0·868	3·474	
Hot	9·15 to 10·15 a.m.	0·644	0·644	1·011
	10·15 to 11·15 a.m.	1·378	1·378	
Sweating	11·15 to 12·15 p.m.	0·044	0·044	
	12·15 to 1·15 p.m.	0·102	0·102	0·498
	1·15 to 2·45 p.m.	1·830	1·220	

CHLORIDE OF SODIUM.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
Night	5 p.m. to 6·30 a.m.	0·576	0·021	0·021
Before fit	6·30 to 7·30 a.m.	0·064	0·064	0·095
	7·30 to 8 a.m.	0·063	0·162	
Cold	8 to 9 a.m.	0·309	0·309	0·381
	9 to 9·15 a.m.	0·167	0·670	
Hot	9·15 to 10·15 a.m.	0·070	0·070	0·090
	10·15 to 11·15 a.m.	0·111	0·111	
	11·15 to 12·15 p.m.	0·019	0·019	0·027
	12·15 to 1·15 p.m.	0·045	0·045	
	1·15 to 2·45 p.m.	0·045	0·030	

QUANTITY OF URINE PASSED.

Night	5 p.m. to 6·30 a.m.	42 c.c.		
Before fit	6·30 to 7·30 a.m.	54 c.c.		
	7·30 to 8 a.m.	60 c.c.		
Cold	8 to 9 a.m.	86 c.c.		
	9 to 9·15 a.m.	172 c.c.		
Hot	9·15 to 10·15 a.m.	26 c.c.		
	10·15 to 11·15 a.m.	52 c.c.		
	11·15 to 12·15 p.m.	13 c.c.		
	12·15 to 1·15 p.m.	38 c.c.		
	1·15 to 2·45 p.m.	50 c.c.		

APRIL 9TH.—UREA.

Immediately before shivering	6·15 to 7·15 a.m.	0·927	0·927	0·927
	7·15 to 8 a.m.	1·081	1·351	1·351
Cold	8 to 9 a.m.	1·089	1·089	1·089
Hot	9 to 10 a.m.	2·152	2·152	1·710
	10 to 11 a.m.	1·268	1·268	
Sweating	11 to 1 p.m.	2·164	1·032	1·129
	1 to 3 p.m.	2·353	1·076	

CHLORIDE OF SODIUM.

Before fit	6·15 to 7·15 a.m.	0·063	0·063	0·063
	7·15 to 8 a.m.	0·124	0·155	0·155
Cold	8 to 9 a.m.	0·231	0·231	0·231
Hot	9 to 10 a.m.	0·561	0·561	0·351
	10 to 11 a.m.	0·140	0·140	
Sweating	11 to 1 p.m.	0·184	0·092	0·078
	1 to 3 p.m.	0·127	0·063	

QUANTITY OF URINE PASSED.

Before fit	6·15 to 7·15 a.m.	35 c.c.		
	7·15 to 8 a.m.	61 c.c.		
Cold	8 to 9 a.m.	55 c.c.		
	9 to 10 a.m.	117 c.c.		
	10 to 11 a.m.	52 c.c.		
	11 to 1 p.m.	44 c.c.		
	1 to 3 p.m.	53 c.c.		

APRIL 10TH.—UREA.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
Before fit	6·30 to 8·15 a.m.	1·420	0·885	0·885
Cold	8·15 to 9·15 a.m.	0·558	0·558	0·558
Hot	9·15 to 10·15 a.m.	1·144	1·144	1·013
	10·15 to 11·15 a.m.	0·883	0·883	
Sweating	11·15 to 2·15 p.m.	0·532	0·177	0·177
	CHLORIDE OF SODIUM.			
Before fit	6·30 to 8·15 a.m.	0·140	0·087	0·087
	8·15 to 9·15 a.m.	Urine lost.		
Hot	9·15 to 10·15 a.m.	0·176	0·176	0·145
	10·15 to 11·15 a.m.	0·115	0·115	
Sweating	11·15 to 2·15 p.m.	0·070	0·032	0·032
	QUANTITY OF URINE PASSED.			
Before fit	6·30 to 8·15 a.m.	50 c.c.		
Cold	8·15 to 9·15 a.m.	20 c.c.		
Hot	9·15 to 10·15 a.m.	44 c.c.		
	10·15 to 11·15 a.m.	32 c.c.		
	11·15 to 2·15 p.m.	22 c.c.		
	APRIL 11TH.—UREA.			
During night ...	2·15 p.m. to 6 a.m.	9·785	0·609	0·609
	6 to 7·30 a.m.	0·921	0·614	0·614
Immediately before fit ...	7·30 to 8 a.m.	0·962	1·924	1·924
Cold	8 to 9 a.m.	1·113	1·113	1·476
	9 to 10 a.m.	1·839	1·839	
Hot	10 to 11 a.m.	0·868	0·868	0·868
Sweating	11 to 1 p.m.	2·090	1·045	1·147
	1 to 2 p.m.	1·356	1·356	
	2 to 4 p.m.	2·090	1·040	
	CHLORIDE OF SODIUM.			
	2·15 p.m. to 6 a.m.	0·139	0·087	0·087
	6 to 7·30 a.m.	0·115	0·076	0·076
Before fit	7·30 to 8 a.m.	0·355	0·355	0·355
Cold	8 to 9 a.m.	0·248	0·248	0·306
	9 to 10 a.m.	0·364	0·364	
Hot	10 to 11 a.m.	0·111	0·111	0·111
Sweating	11 to 1 p.m.	0·114	0·057	0·097
	1 to 2 p.m.	0·136	0·136	
	2 to 4 p.m.	0·119	0·099	

QUANTITY OF URINE PASSED.

Stage.	Hour.	Total Quantity in this time.
Night	2·15 p.m. to 6 a.m.	...
	6 to 7·30 a.m.	24 c.c.
Before fit	7·30 to 8 a.m.	74 c.c.
Cold	8 to 9 a.m.	64 c.c.
	9 to 10 a.m.	76 c.c.
Hot	10 to 11 a.m.	31 c.c.
	11 to 1 p.m.	38 c.c.
	1 to 2 p.m.	57 c.c.
	2 to 4 p.m.	95 c.c.

Conclusions from the previous Facts.

I. *Temperature.*

The temperature during the several fits, amid much general resemblance, presented numerous partial differences, all of which are, however, reducible to order.

(*a*) In every case, the temperature commenced to rise previous to the cold stage,[1] as experienced by the patient, that is to say, before any feeling of cold or illness of any kind. The time that it commenced, previous to the cold stage, varied from an hour and a half to three quarters of an hour, and no connexion between the time and the severity of the fit could be traced. The temperature commenced to rise—

April 6th..1½ hour before any feeling of cold or illness.
„ 7th..1 „ „
„ 8th..1½ „ „
„ 9th..1¼ „ „
„ 10th..1¼ „ „
„ 11th.. ¾ „ „

A close connexion between the severity of the fit and the character of the rise before the cold stage existed, the severity being in proportion to the continuousness of the rise, and also to the extent of each rise. As the fit became less severe, the temperature had a tendency either to oscillate, rise slowly, or remain stationary, and these tendencies

[1] The same fact is noted by Michael, op. cit., p. 43.

increased as the fit decreased in severity, and eventually all combined.

(*b*) During the cold stage the temperature rose throughout, the rise being greater than during any other period; but here also great variations existed, having their counterpart in variations in the severity of the fit.

Thus, in the cold stage, the temperature rises continuously when the fit is severe; then, as the severity lessens, there is a tendency for the rise at the termination of the cold stage to flag, and eventually to become stationary; then the range of each rise becomes less; and lastly, it oscillates. It also appears that the alteration first affects the commencement and termination of the cold stage, an oscillation in its middle indicating a greater diminution of the severity of the fit than at either end.

There was no connexion between the duration of the stage and the severity of the fit, as measured by the temperature. Thus, it lasted—

On the 6th ..... ¾ hour.
„ 7th..... ¾ „
„ 8th.....1¼ „
„ 9th..... ¾ „
„ 10th.....1 „
„ 11th.....2 hours.

The fits were most severe on the 6th and 7th, and declined in severity till the 11th, when the fit was again more severe.

(*c*) During the hot stage the temperature continued to rise, and in its early part closely corresponded to the former periods.

From a careful examination of the charts, it appears that in the severer cases the temperature in the hot stage ran up at once to its acme, and had a tendency to remain permanent; the permanency not, however, being obtained at once nor retained throughout, the temperature oscillating both at the commencement and termination. In less severe cases it ran up slowly, and did not remain stationary, and when least severe, oscillated in its rise.

The temperature in those fits becoming less severe first lost its permanency, and next the temperature rose to a less extent

and again became permanent; then, when they became still less severe, it rose to the same extent, but again lost its permanency. Again, in those cases where the temperature remained permanent, the hot stage ended at the termination of the permanent period in an oscillation, these cases being the severest at their own temperature. In all other cases the temperature fell before the sweating stage commenced, ending in an oscillation, and the less severe the fit the greater was the portion of the hot stage occupied by the fall of the temperature. Also the fit was less severe, and the fall greater, when the temperature in falling every now and then stood still for some time, and was still less severe when it oscillated.

(*d*) The temperature continued to fall through the sweating stage, and was often a long time before it reached the point from which it started. The temperature fell most rapidly in those cases in which the oscillations occurred, and least rapidly where the fall was continuous. Towards the termination of the sweating stage a rise occurred to a slight extent (perhaps followed by oscillations) in four of the six fits. Thus, on April 6th, sweating commenced at 11·15, when the thermometer marked 103⅕°; at 2·15 the temperature had fallen to 100°; it then rose to 101° at 2·30, to 101⅖° at 2·45; then fell to 100⅕° and 100° at 3 and 3·15; then rose to 100⅕° at 3·30, and then finally fell regularly to 97⅗°, a point not reached till seven o'clock. The charts show, at a glance, these slight but perfectly definite rises at this late period of the sweating stage.

II. *Urea.*

As the type of the disease in this man was quotidian, there was no opportunity of comparing the amount of urea on a fever and on a fever-free *day*. But the amount of urea passed in the fever-free *hours* was decidedly much smaller than might have been anticipated. He excreted only 0·650 grm. on an average per hour in the apyrectic period, which would give in twenty-four hours 15·600 grms., had the excretion remained at the same amount during

the whole day. Now, a man of the same weight, between twenty and forty years of age, on a good diet, as this man was, would have secreted 32 grms. in the twenty-four hours, if he passed the average amount. Our patient was, however, older (fifty-nine), and would, no doubt, form less urea than a man at a more vigorous period of life. But it can hardly be supposed that the amount would be reduced so low as 15½ grms. in the twenty-four hours by this difference of age. It may, therefore, be concluded that, in accordance with Redenbacher's observations, the excretion of urea in the fever-free period was below the healthy amount.

The observations made by Traube, Redenbacher, and Hammond on the increase of urea during the cold and hot stages are entirely confirmed by this case, but a more minute statement of the kind and amount of the increase can now be given.

It must, however, be premised, that the only obvious causes of the increase of the urea in this case during the fit are, either the food taken at breakfast at 5 a.m., the fluid drunk during the fit, or the fit itself. No other known causes existed which could have had the effect of increasing the urea. The following objections to the idea of the food being the cause may be urged. That the amount of food was not great, and that the increase in the urea was far larger than has yet been noted after even the heaviest meal. For example, on the 7th, the amount per hour rose from 0·684 grm. to 2·176, being an increase of more than 200 per cent.; on the 8th, from 0·654 to 3·474, being an increase of nearly 500 per cent.; on the 9th and 10th, when the fits were slighter, the increase was less marked, though still considerable; while on the 11th, when the fit was severe, it was again 200 per cent. Such an amount is greater than has yet been found after an ordinary amount of food.[1] The time, however, at which the urea increases after food accords with our case; for, augmenting even

[1] Dr. Parkes has noted in one person without fever a rise from 0·665 grm. in a fasting hour to 1·554 grm. in a food hour, but this was after a hearty dinner.

during the first hour after food,[1] it attains its maximum sometimes in the third hour;[2] sometimes, however, not till the seventh hour; usually, however, it reaches its maximum at the fourth hour.

Now, in this case food was taken between 5 and 6 a.m., and the maximum amount of urea secreted was during the cold stage, from 8 to 9·30 a.m., or in the fourth and fifth hours. But it will probably be conceded, after it has been shown how closely the amount of the urea was associated with the variation in the temperature, that its increase in the fourth and fifth hours after food was merely a coincidence, and was not owing to the very moderate breakfast, but to the fact of the highest temperature occurring at this time. On one day, moreover, he took no food, having no appetite, and on this day the usual increase occurred. Again, after the fits were stopped by the quinine, the food was given him as usual, and the urine being collected on the same day, in the same way, comparatively little rise took place in the urea. These two last points, I conceive, set the question quite at rest.

With respect to the amount of fluid drunk, this could have no effect on the urea, as very little fluid was taken till after the time when the urea had commenced to rise. The urea, moreover, reached its maximum often at the termination of the cold stage, whilst he seldom drank anything between his breakfast and the hot stage. I believe, then, that I am justified in concluding that the rise in the amount of urea was not owing either to food or liquid. It must, therefore, have been owing to the fit.

The urea begins to increase in amount *before* the cold stage, as judged of by the first feeling of shivering, in four of the five fits. Thus, it rose—

On the 7th, from 0·666 to 1·361 per hour.
„ 8th, from 0·654 to 1·425 „
„ 9th, from 0·927 to 1·351 „
„ 11th, from 0·614 to 1·924 „

[1] Voit, quoted by Meissner, "Report on Phys. for 1857," in Henle's 'Zeitschrift,' p. 352.

[2] Becker, Henle's 'Zeitschrift,' 1855, p. 549.

The time it commenced to rise before the subjective fit varied. Thus—

On the 7th.... ¾ hour before.
„ 8th....1½ „
„ 9th....1¾ „
„ 11th.... ½ „

It often began to increase, indeed, even before the temperature began to rise. Thus, as the temperature on the 7th rose before the fit ¾ of an hour, on the 8th 1¼, on the 9th ¾, the urea on those days commenced to rise previous to any similar change in the temperature.

Great apparent irregularity existed in the rise of urea, but a close correspondence is observed between these variations and similar ones in the temperature.

The alteration in the above case of urea and the temperature did not always exactly agree in time, though this was the rule, but sometimes the alteration in the temperature did not occur till after the alteration in the urea; the reverse never happened.

The quantity of urea continued to increase, and reached its highest point either at the termination of the cold stage, as on the 8th and 11th, or at the commencement of the hot, as on the 6th, 9th, 10th, and 12th. It then began to fall in quantity, slowly at first. On the 6th and 10th the temperature and urea commenced to fall simultaneously. On the 7th, 8th, 9th, and 11th, the temperature continued to rise, notwithstanding the fall in the urea. Up to the period when the urea commenced to decrease, the temperature rose rapidly, each rise being extensive, but after that, at the point where the urea commenced to fall, the temperature either oscillated, or every rise remained stationary for a short time, the rise being always slow and by small additions.

The urea continued to fall slowly from the end of the cold, or from the commencement of the hot stage, till the sweating stage began; and the temperature corresponding in time to the latter part of this slow decrease, after reaching its height, fell also slowly and slightly.

From the following table it appears that no close correspondence existed between the fall in the temperature towards the close of the hot stage and the decrease in the amount of urea.

Date.	Fall in Urea.	Fall in Temperature in same time.	Temperature oscillated.	Tendency to be stationary.	Temperature fell continuously.	Fall of Urea reduced to quantity for 1° Fahr.
6th	0·268	$\frac{1}{8}$°		—		1·346
7th	0·830	$1\frac{3}{8}$°			—	0·533
8th	1·452	$\frac{3}{8}$°			—	...
9th	0·884	1°			—	0·884
10th	0·261	$\frac{3}{8}$°		—		0·435
11th	1·000	$2\frac{3}{8}$°	—			0·357

In the first column the date is given; in the second the fall in the amount of urea, corresponding in time to the fall in the temperature, from its highest point to the commencement of the sweating stage, is given; next, the number of degrees the temperature fell in the same time; then, in three columns, the character of the fall is given; and in the last column the fall in the amount of urea is reduced to the quantity corresponding to each degree. But though there is no intimate connexion, still the greater the fall in the temperature the greater is the decrease in the quantity of urea. At the same time the character of the fall varied; thus, sometimes the temperature showed a tendency to remain stationary, then the urea fell but little, as on the 6th and 10th. On other days the temperature fell gradually, and then the fall in urea rather increased. The greatest fall occurred on those days when the temperature oscillated greatly. Thus, the least decrease in urea corresponded to that temperature which has a tendency to remain stationary, more when it fell slowly but continuously, and much the most when it oscillated, as on the 11th; and it is

possible to judge of the rapidity of the decrease of the urea by the character of the temperature.

The temperature has been shown to fall either at the close of the hot or the commencement of the sweating stage. A similar fall occurred in the urea, and the amount of the decrease of urea corresponded to the length of the oscillation, and the subsequent rise in the urea corresponded to the rise in the oscillation, though the whole oscillation always occurred in an hour previous to that in which the subsequent rise in the urea occurred.

Date.	Fall in Temperature.	Fall in Urea.	Rise in Temperature.	Rise in Urea.
6th	1⅝°	1·820 to 1·289	1⅝°	1·289 to 1·620
7th	1°	1·287 to 0·838	1°	0·838 to 1·222
8th	1⅝°	3·474 to 0·644	⅝°	0·644 to 1·378
11th	1⅝°	1·839 to 1·045	⅛°	1·045 to 1·356

A slight rise, however, took place at the same period, even when the temperature remained permanent, but then the increase was small in amount.

On the 9th, the temperature fell, but did not oscillate; the fall in the urea was not so great as when the temperature oscillated.

A relationship in the latter part of the sweating stage between the variation in the urea and temperature existed, corresponding to what is stated above. Thus, on the 6th, after the usual oscillation, the temperature had a tendency to fall, and then to remain stationary for some time, and again to fall and remain a second time stationary. During this time the urea fell in quantity slowly. The temperature then took a great fall, and oscillated greatly, and at the same time the fall in urea was very great.

On the 7th the temperature fell slowly, and often remained stationary; at the same time the urea fell slowly, and, like the temperature, did not reach the normal amount till late in the afternoon.

The same close correspondence exists between the other cases.

In order to test still further the relationship between the urea and the temperature, the following method was adopted. The total amounts of urea excreted during the fit, the quantity excreted during the rise and fall of temperature, during the period immediately before the fit, and during the cold and hot stages, respectively, have been added up and divided by the number of degrees the temperature rose or fell in the corresponding periods. By this means the amounts of the urea secreted during the whole and each of the divisions of the fit were reduced to a common standard, and could thus be compared more easily with one another.

Table[1] *to show the amount of Urea corresponding to each degree of Fahrenheit of abnormal heat.*

Date.	Entire fit. The amount of urea for each degree of abnormal heat.	Rise in the temperature taken, and the amount of urea to each degree.	Fall in the temperature and the urea to each degree.	The quantity of urea to each degree of the temperature before the fit.	The same in the cold stage.	The same in the hot stage.
6th	1·557	0·626	0·930	...	0·339	3·560
7th	1·413	0·615	0·798	0·972	0·391	1·609
8th	1·508	0·903	0·722	2·163	0·735	1·444
9th	1·460	0·675	0·710	0·566	0·246	2·793
0th	1·298	0·718	0·746	0·657	0·617	1·381
11th	1·781	0·712	1·280	0·601	0·590	0·868

[1] In this table each of the quantities of urea correspond to 1° of temperature. In the first column the date is given; in the second, the whole amount of urea passed during the entire fit (the fit being considered to

On comparing the different days, the closest correspondence existed when the whole fit was taken. The extreme in column two being taken, the difference only amounted to one fifth of a degree. The rise and fall also closely corresponded. The extreme again being taken, a difference amounting to one third of a degree was found.

As the duration of the fit might have varied greatly, and so the above mode of comparison of the different days one with the other have been invalidated, the following tables have been worked out.

The first shows the duration of the fit on each day.

6th.....	7¼	hours' duration.
7th.....	8	„
8th.....	8¼	„
9th.....	7¾	„
10th.....	8¼	„
11th.....	7½	„

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commence with the rise of the temperature and the increase of the urea and chloride of sodium). In the third column the rise (in the temperature) only is taken, and when the temperature remained stationary at its highest point for some time the time of the rise is calculated up to the middle of the stationary part. In the next column the fall, calculated in the same way, is given. Then the cold and hot stages are given separately.

The amount of urea varied in this patient; for the night urine varied on one day greatly from the quantity passed during a similar period on other nights. This day, the 9th (the urine of the night before not having been saved), the amount of urea corresponding to each degree was greatly in excess; but on assuming that the quantity passed during the first hour 0·900 grm.) was the quantity normal to him for that day, and so deducting the excess, a close correspondence to the other days resulted. Thus, when the whole amount of urea was calculated as it stood, to each degree 2·043 grms. were found to correspond; but when the additional 300 grms. were deducted, then the quantity per degree was 1·460; and throughout all the different comparisons it will be then found closely to agree. On the 10th, again, the normal excretion was below par (0·170 grm. per hour), which also prevented any comparisons from being made. When this was raised to 0·600 grm., then a much closer correspondence to the other days was found.

Another calculation must now be made. On three days the urine was collected through the previous night; taking that hourly amount, and assuming that to be the amount normal to the man through that day, if there had been no disturbing influences, and deducting that quantity from the quantity passed hourly through the fit, the increase only is obtained, and this divided by the number of degrees the temperature rose, the comparison is then found to be very close.

April 7th.....0·638 urea to each degree of abnormal heat.
„ 8th.....0·618 „ „
„ 11th.....0·624 „ „

If this table be compared with the former one, the comparison will be found to be even much closer. Unfortunately, the amount during the night was not determined except on these three days, so that this mode of calculation is not applicable to all the fits.

On looking at the second column of the table given at page 377, where the total amounts are compared, it is found that on the 6th, 8th, and 11th, the quantity in excess is somewhat over other days. The temperature on the 6th and 8th remained permanent after reaching its highest point. Thus, on the 6th it remained at 105° for two hours and a half; on the 8th at $103\frac{1}{5}$° for one hour and a quarter; but when the amount passed during these permanent periods is deducted, then the urea on the 6th fell to 1·277 grm., and on the 8th to 1·366 grm., bringing each on a level with the 10th. On the 11th, the increase in urea occurred entirely in the fourth column, that is, during the fall of the temperature, the amount passed during the rise corresponding closely to the other days; and on examining the sweating stage on the 11th, there is found a great and sudden increase at its very termination, and this excessive quantity being replaced by the quantity passed on other days at the same period, the amount corresponding to each degree was 1·510, closely coinciding with the other days.

That the increase on the 6th and 8th was due to the above is rendered probable by the following statement: The whole quantity of urea passed on the 6th, during the stationary period of two hours and a half, was 1·957 grm. On the 8th, for one hour and a quarter, the quantity was 1·155. When compared to equal time, that is, the two hours and a half reduced to one hour and a quarter, then, on the 6th, the amount of urea was 0·978, corresponding closely to the quantity passed in the same time on the 8th. From this it would appear that the same amount of urea corresponds to 1°, when the temperature is permanent, whether it be at a high temperature or not, whilst the second table shows that *every degree in the hot stage corresponds to a much greater amount of urea than in the cold or any other stage, whilst the temperature is rising;* thus showing, that the higher the temperature, the more urea corresponds with each rise of a degree than formerly, and thus accounting in some measure for the greater quantity of urea to each degree, when the whole fit is compared, in the more severe fits.

The very close correspondence between the temperature and the urea seems to be thus placed beyond doubt; in fact, the one may be calculated from the other. An example or two will illustrate this.

Thus, on—

April 6th, the amount of urea excreted during the fit was 11·097 grammes; taking 1·503 gramme as the average quantity (all the degrees being taken) corresponding to each degree, this gives a rise of 7°, or a little over; the actual rise was from $98\frac{1}{5}$ to 105, or $6\frac{4}{5}$°.

April 7th, the total amount of urea during the fit was 10·635 grammes. The rise in the temperature here should be 7°; the actual rise was $6\frac{4}{5}$°.

April 8th, the total amount of urea excreted was 9·353 grammes, which would imply a rise in the temperature of 6°; the actual rise was from $97\frac{4}{5}$ to $103\frac{4}{5}$, being exactly 6°.

III. *Chloride of Sodium.*

The chloride of sodium, to a very great extent, agreed with the urea in its relationship to the temperature. Traube's statement on this point is therefore confirmed. For the most part, the remarks above made on the urea hold good for the chloride of sodium; still some differences occurred.

On the 8th, the urea between 7·30 and 8 a.m. remained stationary, whilst the chloride of sodium rose continuously.

9th.—Between 8 and 9 a.m. the urea fell, the temperature corresponding, but the chloride of sodium rose to double its quantity.

Again, during the decline of the fit, they differed on the 8th and 9th; the urea at its termination rose somewhat, whilst the chloride of sodium continued to fall.

The following table corresponds to one given for the urea, and shows the intimate connexion between the severity of the fit and the amount of chloride.

Date.	Entire fit. The amount of chloride of sodium for each degree.	Rise in the temperature taken, and the amount of chloride to each degree.	Fall in the temperature taken, and the amount of chloride to each degree.	The quantity of chloride to each degree of the temperature during the cold stage.	The same during the hot stage.	The same during the period before the cold stage.
6th	0·195	0·085	0·086	0·027	0·293	...
7th	0·225	0·159	0·066	0·085	0·259	0·640
8th	0·171	0·107	0·078	0·131	0·162	0·134
9th	0·241	0·150	0·062	0·175	0·702	0·103
10th			Lost.			
11th	0·180	0·149	0·041	0·102	0·176	0·785

Here, as occurred in the urea, the patient on two days

passed a different quantity normally from what he had on former days; so that on one day, the 8th, it was necessary to add 0·022 grm. to each hour to bring it up to the other days. On the 11th it was necessary to subtract 0·044 grm.

On the 6th the urine was not collected before the commencement of the cold stage, so that the amount for each degree is less than it would otherwise have been in the third and fourth columns.

When the extremes in the third column were compared, a difference corresponding to one third of a degree of temperature occurred. When the fourth column was compared, excluding the 6th, a difference of one third of a degree occurred. In the fifth column, a difference corresponding to less than one third occurred, excluding the 11th, on which day the chloride fell to its normal amount before the fit had ended.

In the cold stage an enormous difference was found.

In the hot the difference corresponded to half of a degree.

Beyond these above differences, all that has been said regarding the urea applies equally to the chloride of sodium.

IV. *Water of the Urine.*

In rising and falling in quantity, the water has a very close correspondence to the urea, though there is very little proportion between the different rises and falls in the two. On the 6th, 8th, and 9th, they corresponded in rising and falling at the same periods. On 11th and 7th the quantity of urine in the hot stage falls so considerably, that in the commencement of the sweating stage a slight rise occurs, this being followed, however, by a decided fall during the second hour.

The quantity of urine thus corresponding to the urea, must, like it, correspond somewhat to the variations in the temperature. That the quantity stands in close relation to the intensity of the fit is seen from the following table.

Date.[1]	Quantity of urine in the fit.	Number of degrees temperature rose.	Quantity of urine to each degree.
6th	608 c.c.	5$\frac{1}{5}$	117 c.c.
7th	411 c.c.	5$\frac{4}{5}$	71 c.c.
8th	449 c.c.	6$\frac{1}{5}$	72 c.c.
9th	382 c.c.	5$\frac{2}{5}$	71 c.c.
11th	435 c.c.	5$\frac{4}{5}$	75 c.c.

It has been shown that some correspondence exists between the oscillation of temperature at the commencement of the sweating stage and the fall and subsequent rise in the amount of urea. The following table shows the relationship between the same oscillation and the fall in the amount of urine passed.

On the 6th and 11th it has been said that the amount of urine secreted was so small during the hot stage that it rose somewhat at the commencement of the sweating, so that no comparison can be given on those days between the fall of the oscillation and the quantity of urine. In the next table, in the first column, as usual, the day of the month is given; in the second, the fall of the temperature in the oscillation; in the third, the fall in the amount of urine; in the fourth, the amount reduced to a degree; in the fifth column, the rise of the temperature in the oscillation is given; in the sixth, the rise in the amount of urine in the hour subsequent; and in the seventh, the quantity is reduced to that corresponding to a degree:

[1] In the first column the date is given. In the second, the total quantity of urine passed during the fit. In the third, the number of degrees the temperature rose during the fit. In the fourth, the number of cubic centimetres corresponding to each degree. Excluding the first day, a very close correspondence existed.

6th	Urine rose		...	1½°	0·030	0·020
7th	1°	0·030	0·030	1°	0·017	0·017
8th	1½°	0·039	0·025	½°	0·025	0·030
11th	Urine rose		...	½°	0·007	0·035

From this limited table, no great correspondence can be traced.

But though the urea, chloride of sodium, and water, thus constantly show a close correspondence to the temperature, the relative amount of rise was different in these three ingredients.

This indeed was shown well during the analysis, in which 10 c.c. of urine were taken; the amount of mercury solution required for the amount of urea varied greatly from hour to hour, showing no regularity of rise; whilst, on the other hand, the amount required in testing for the chloride gradually rose and then gradually sank, often again rising somewhat at the very close of the fit. Thus, to take the 7th of April:

UREA.

Hour.	Amount of mercury solution required for 100 pints of urine.	Same for NaCl.	Hourly amount of water passed.
7 a.m.	298 c.c.	48 c.c.	25 c.c.
8 a.m.	198 c.c.	144 c.c.	80 c.c.
8·30 a.m.	188 c.c.	135 c.c.	136 c.c.
10·30 a.m.	224 c.c.	90 c.c.	62 c.c.
12 noon.	272 c.c.	45 c.c.	33 c.c.
1 p.m.	268 c.c.	45 c.c.	50 c.c.
3 p.m.	248 c.c.	36 c.c.	50 c.c.
5 p.m.	258 c.c.	18 c.c.	28 c.c.

The urea varies in its proportion to the water; thus, during excessive diuresis, the per-centage amount falls, whilst, on the other hand, the per-centage amount of chloride even then increases, up to the time the greatest amount is poured out, then it as steadily falls, even when the water fluctuates

greatly. These remarks apply to the per-centage of chloride of sodium in the urine.

It appears that the amount of urea undergoes an increase definite in amount, independent of the water. The chloride of sodium also undergoes a definite increase, which, also, is independent of the amount of water; but the water being increased, the same per-centage of chloride is poured out as would have been the case if a smaller amount of water had been voided, the per-centage not being lowered by an excess of water, as is the case with urea.

The chloride has thus a tendency to rise and fall steadily, not observing the various alterations corresponding to temperature that the urea does, but the water, corresponding to the urea in this respect, causes variations of the same character in the total amount of chloride poured out.

The time of greatest per-centage excretion of chloride of sodium does not always correspond to the greatest hourly excretion.

Thus, on April 6th, the greatest amount of saturation was at 8 a.m., whilst the greatest hourly excretion was at 8·30, when the water was at its greatest amount.

7th.—The hourly excretion and the per-centage amount agreed in the time at which they occurred, and so also did the water.

8th.—The same occurred on this day.

9th and 10th.—The same occurred also on these days.

11th.—The per-centage amount of chloride was greatest at 9 a.m., whilst the hourly amount excreted was greatest at 10, the water being most abundant during the last period.

The urea, on the other hand, often decreased in per-centage during the fit, especially if the increase in the water was great, its highest per-centage amount corresponding to the lesser amount of water. Thus, on April 6th, the per-centage amount decreased through the entire fit. The decrease was, however, much more gradual at its commencement.

7th.—The greatest per-centage amount was at 7 a.m.; the amount of water being 25 c.c. for the hour. The water

then rose to 80 c.c., and the per-centage amount of urea fell; the amount of mercury solution required for 10 c.c. of urine fell from 29 c.c. to 19 c.c. The water then again rose to 136 c.c., and the solution of mercury required fell in amount to 18 c.c. The water next fell to 62 c.c., and the solution of mercury rose to 22·6 c.c. The water again fell to 33 c.c., and the amount of the solution required rose to 27·4 c.c. The water next rose to 50 c.c., and the solution required fell to 26·4 c.c. The water then remained at 50 c.c., but the solution fell to 24 c.c. Then the water fell to 28 c.c., and the quantity of the solution rose to 25 c.c.[1]

From these facts it is evident that both the urea and chloride increase, independent of the influence of the amount of water; that any increase in the latter does not modify the aggregate amount of urea, but that it does that of the chloride.

No connexion exists between the quantity of urine passed during the entire fit and the amount of water drunk.

This is seen in the following table:[2]

Date.	Water drunk.	Urine passed.
6th	2135 c.c.	648 c.c.
7th	1335 „	550 „
8th	1050 „	428 „
9th	500 „	499 „

The urine was passed much more equally as regards the different periods than the water drunk, for the entire quantity was mostly drunk during the hot stage only.

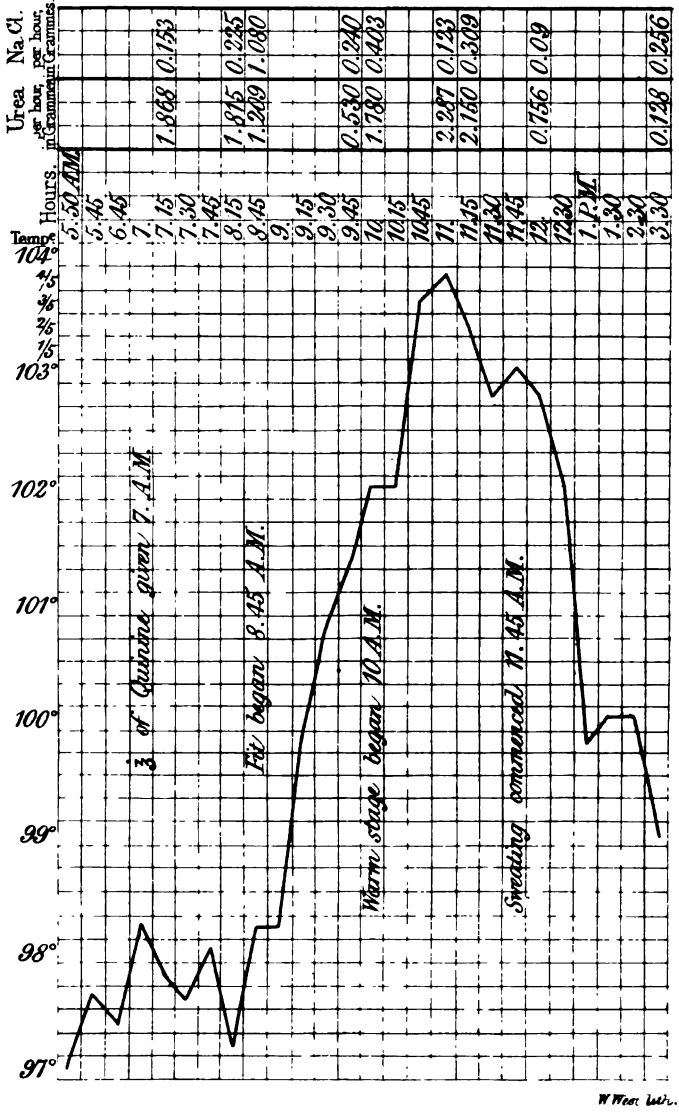
Thus, the urine, urea, and chloride of sodium, were independent, to a large extent, of the quantity drunk,

[1] Every c.c. of the mercury solution corresponds to 10 milligrammes of urea.

[2] As usual, the date is given in the first column; in the second, the quantity of water drunk during the entire fit, and in the third, the amount of urine passed during the entire fit, are given.

APRIL 12TH

Phenomena of a Fit modified by Quinine.



though some connexion appeared to exist, as, when the patient drank copiously, the next time the urine was collected it was found to be somewhat in excess; but this might be a mere coincidence.

TABLE.

APRIL 12.—UREA.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
Before fit	6·15 to 7·15	1·868	1·868	1·630
	7·15 to 8·15	1·815	1·815	
	8·15 to 8·45	0·604	1·209	
Cold	8·45 to 9·45	0·530	0·530	2·310
	9·45 to 10	0·445	1·780	
Hot	10 to 11	2·287	2·287	2·219
	11 to 11·30	1·075	2·150	
Sweating	11·30 to 12·30	0·756	0·756	0·442
	12·30 to 3·30	0·384	0·128	
	CHLORIDE OF SODIUM.			
Before fit	6·15 to 7·15	0·153	0·153	0·153
	7·15 to 8·15	0·225	0·225	0·225
	8·15 to 8·45	0·540	1·080	1·080
Cold	8·45 to 9·45	0·240	0·240	0·321
	9·45 to 10	0·100	0·403	
Hot	10 to 11	0·625	0·625	0·467
	11 to 11·30	0·154	0·309	
	11·30 to 12·30	0·090	0·090	0·173
	12·30 to 3·30	0·708	0·256	
	QUANTITY OF URINE PASSED.			
Before fit	6 to 7·15	73 c.c.		
	7·15 to 8·15	75 c.c.		
	8·15 to 8·45	54 c.c.		
Cold	8·45 to 9·45	25 c.c.		
	9·45 to 10	84 c.c.		
Hot	10 to 11	125 c.c.		
	11 to 11·30	86 c.c.		
	11·30 to 12·30	32 c.c.		
	12·30 to 3·30	128 c.c.		

On the 12th, everything being conducted in the same manner, upon the temperature commencing to rise, the patient was given ℈j of quinine, which caused the tempera-

ture to fall again. It continued to fall for half an hour, and then began to rise; the cold stage came on an hour later than on the previous day. The fit, however, was afterwards as severe as usual. The effects on the urea corresponding to this fall cannot be ascertained, as, for some reason, the amount secreted was three times as great as on previous days, for the first two hours it was collected; it then fell somewhat, but continued high throughout the fit, the quantity to each degree being 0·980 grammes.

The temperature obeyed all the rules laid down previously—rising to the commencement of the cold stage; then remaining permanent for half an hour; then rising through the whole of the cold stage, the latter being shorter than the former; and just at the commencement of the hot stage, it again remained stationary for a quarter of an hour; then rose, but began to fall before the sweating stage commenced, and at that point oscillated, the oscillation being small; it then fell rapidly.

The urea corresponded also to these variations, after the manner described. Thus, it fell where the temperature remained stationary, but the fall was out of proportion to the alteration in the temperature, being very great. Possibly, this was due to the quinine. It then rose rapidly, reaching its highest point at the same time as the temperature. It fell slowly during the latter part of the hot stage, then fell greatly during the oscillation at the commencement of the sweating stage; but as the urea did not subsequently rise, the rise in the oscillation of the temperature was only one fifth; then the urea fell greatly.

The chloride of sodium differed somewhat from the urea at the commencement and termination. Thus, though an unusual quantity was passed in the first two hours, like the urea, still it did not fall when the temperature began to rise a second time, but continued rising throughout; just before the cold stage it reached its highest point, arriving at the very unusual quantity of 1·080 grm.

After this it corresponded in its variations to the urea, except in the second hour of the sweating stage, when, instead of continuing to fall, it rose considerably. The rise in the hour subsequent to the oscillation in the temperature was very extensive.

Considering the fit to have commenced on this day, at 7·15 a.m., the amount for each degree was 0·396, a larger quantity (as is also the case with the urea) than occurred on other days; but as the amount passed for some hours before the fit was unusually large, it is probable that the quantity normal for that day was in excess.

The water in its variations corresponded to the urea, except at its very termination (that is to say, the second hour of the sweating stage), when it rose considerably.

The amount for each degree was 105 c.c. It is rendered most highly probable that the reason for the amount for each degree being greater than usual in urea, salt, and water, is, that the normal quantity was in excess on this day, for it is found that the increase in each is proportionate.

The quantity he drank was not taken down on this day. Thus the only influence that can be ascribed to the quinine is the lowering of the temperature and the postponement of the fit.

Examination of the Urine on April 13*th*, ℈ij *of Quinine having been taken the day before, the fit being entirely absent.*

On the 13th no fit occurred. The patient took, without leave, another scruple of quinine the evening before. The temperature remained at 97° the entire day, with very slight variations, never amounting to more than $\frac{2}{5}$°. But, notwithstanding this equality of temperature, the urea and chloride of sodium underwent an increase. The urine was collected through the night.

UREA.		CHLORIDE OF SODIUM.	
Hour.	Quantity per hour.	Hour.	Quantity per hour.
	Grammes.		Grammes.
3·30 p.m. to 6·30 a.m.	0·610	3·30 p.m. to 6·30 a.m.	0·057
6·30 to 7·30 a.m.	1·340	6·30 to 7·30 a.m.	0·060
7·30 to 8·30 a.m.	1·122	7·30 to 8·30 a.m.	0·082
8·30 to 9·30 a.m.	1·150	8·30 to 9·30 a.m.	0·096
9·30 to 10·30 a.m.	1·412	9·30 to 10·30 a.m.	0·138
10·30 to 11·30 a.m.	0·803	10·30 to 11·30 a.m.	0·086
11·30 to 12·30 p.m.	0·999	11·30 to 12·30 p.m.	0·091
12·30 to 1·30 p.m.	0·606	12·30 to 1·30 p.m.	0·027

Thus, though the patient experienced no change as regards his sensations, and his temperature remained the same throughout the day, *a similar change to that occurring on the previous days was observed in the urea and chloride of sodium.* The fit, as judged by the urine, commenced at 6·30 a.m. The urea then fell somewhat during the next hour, rising again steadily up to 10·30 a.m., when it had reached its highest point. It then fell greatly, and this was followed by a slight rise, and then again fell to the amount normal to this man. The fall, immediately after the first rise, probably corresponded to the termination of the period prior to the cold or the commencement of the cold stage; and had the temperature risen, it would probably at this point have either oscillated or remained permanent for some time.

The next great fall, at 11·30 a.m., corresponded to the sweating stage, and would have been marked in the temperature by a great fall, and then, probably, the temperature would have remained stationary for half an hour, and not have risen, as the subsequent rise in the urea is small in amount. The urea after this small rise again falls to the normal amount.

The chloride also strictly followed the principles laid down, for, as has been shown, it has a tendency to rise continuously, an equal amount of urine being taken each hour, though, if the urine be much altered in

quantity, this salt also falls and rises. Thus, the chloride rises continuously, and is marked by no fall after the first hour, but, corresponding to the great fall of urea at 11·30, it also falls and again rises slightly in the next hour, and then again falls to the amount normal.

The urea and chloride increase *pari passu*, and each reaches its height at the same hour.

The amount of urine, again, corresponded to the urea and chloride of sodium; it fell slightly after the first hour, then rose, reaching its highest point at the same time as the urea; it then fell, but again rose somewhat, and then permanently fell.

3·30 p.m.	to	6·30 a.m.	40 c.c.
6·30 a.m.	to	7·30 „	50 „
7·30 „		8·30 „	46 „
8·30 „		9·30 „	46 „
9·30 „		10·30 „	66 „
10·30 „		11·30 „	41 „
11·30 „		12·30 p.m.	51 „
12·30 p.m.	to	1·30 „	30 „

Taking the mean quantity of grammes corresponding to a single degree on other days, the increase of urea would correspond to a rise of 5¼° in temperature. Thus, as far as the urea, &c., are concerned, the fit was as severe as on previous days.

The urine was again collected hourly during the 14th.

Here the urea and chloride of sodium were again in great excess during the whole day, and also during the night previous.

Hour.	Urea per hour.	Chloride per hour.	Water per hour.
1·30 p.m. to 6 a.m.	1·620	0·168	85 c.c.
6 to 7 a.m.	2·715	0·585	150 c.c.
7 to 8 a.m.	1·332	0·405	90 c.c.
8 to 9 a.m.	2·545	0·877	172 c.c.
9 to 10·30 a.m.	1·106	0·320	67 c.c.
10·30 to 11·30 a.m.	1·107	0·421	78 c.c.
11·30 a.m. to 1·30 p.m.	1·118	0·312	60 c.c.
1·30 to 3·30 p.m.	1·180	0·254	77 c.c.

On the second and fourth hours the urea rose to nearly double the quantity of other hours. After the second rise it remained stationary for two hours, and then rose. This corresponds to some extent with the urea the day before; but after the second rise the fall was not so great, and the subsequent rise was very small in amount.

The chloride more closely resembled the day before. Like the urea of the same day, it rose twice, but the last rise exceeded the first, and was followed by a considerable fall, and this again by a rise of some extent, and then it again fell. The second rise and fall corresponding to the commencement of the sweating stage, the first fall probably would have corresponded (had the temperature risen) to an oscillation or a permanency at the termination of the period prior to the cold stage.

The water closely corresponded to the chloride.

Detracting 1·000 gramme from each hour, that being the necessary quantity to reduce the urea in the night urine to the amount normal to this man, and again, as on the day before, calculating what height the temperature would have reached had it risen, it is found to be 3°.

This fit was therefore less severe than that on the previous day. It thus appears that *variations in the urea and chloride of sodium continue to occur at those periods when, if the fit had continued, the temperature would have risen.* The same fact has been noted by Redenbacher; and it would show that the cure of the fit by quinine, in this man, was followed by a much larger excretion of urea during what would have been the apyrectic hours than had been noted at the corresponding time when he was suffering from the disease.

Case 2.—*Tertian ague.*—The following case of ague occurred in a boy, æt. 19, strong, and in every respect healthy with exception of the attack of ague. The boy had not been out of London for twelve months before, but at that time came over from Dantzig, having been there for some months.

He was admitted into the hospital with the second fit, which was very slightly marked, the fit increasing subsequently in severity till he took four grains of quinine every two hours, which prevented its continuance. It was tertian in type.

Everything was conducted exactly after the manner of the former case. The urine, however, was collected (on one day only) by the stage, and not by the hour. His pulse was taken each quarter of an hour, with the temperature.

The two following charts of the temperature will be found to correspond closely with those given before, the correspondence holding mostly with the severer fits. In the first, the temperature rises slowly up to the cold stage; it then rises rapidly, running up continuously to the commencement of the hot stage, at which point it remains stationary for half an hour; it then runs up to its highest point, remains stationary for a short time, and sinks till the sweating stage commences, when, instead of oscillating, it remains stationary for half an hour, then falls and oscillates, but remains high even at 9 p.m., thus differing from the former and subsequent tables.

This further illustrates the slow rise of the stage previous to the cold one; then its rapid rise, the rise being here also greater than at any other period; also its tendency to be affected first at either termination as the fit becomes less severe. It shows that the temperature falls before the commencement of the sweating stage, and that at this point it either oscillates or remains stationary; and in the severe fit, that the temperature has a tendency to remain stationary at its highest point.

The next chart (that of April 4th) shows the temperature on the day the fit was severer, and consequently we find indications of this in the character and fall of the temperature. It shows at the commencement of the cold stage a tendency to be stationary, but after this it runs up continuously with no permanent period at the termination

of the cold stage, though an approximation is seen in the rise at this point being less than the previous ones. It then remains permanent for some time, and then very slowly sinks, remaining permanent for half an hour just where the sweating commences; the temperature here also falling before the sweating stage commenced. Thus, the continuous rise, the permanency when at its height, and the slow and gradual fall, all indicate the severity of the fit, besides the great height to which the temperature reaches.

The urea, here only estimated for each stage, corresponds to the results before stated. Taking the average amount per hour in the sweating stage as the nearest approach to the quantity normal to the boy, it is found that there is an increase in the period previous to the fit; this continues increasing, is highest during the cold stage, then falls somewhat in the hot, and again falls still lower in the sweating.[1]

The same is exactly the case with the chloride of sodium and water.[2]

The patient drank—

At breakfast	285 c.c.
Before cold stage	200 „
Cold stage	200 „
Hot stage	700 „
Sweating stage	500 „

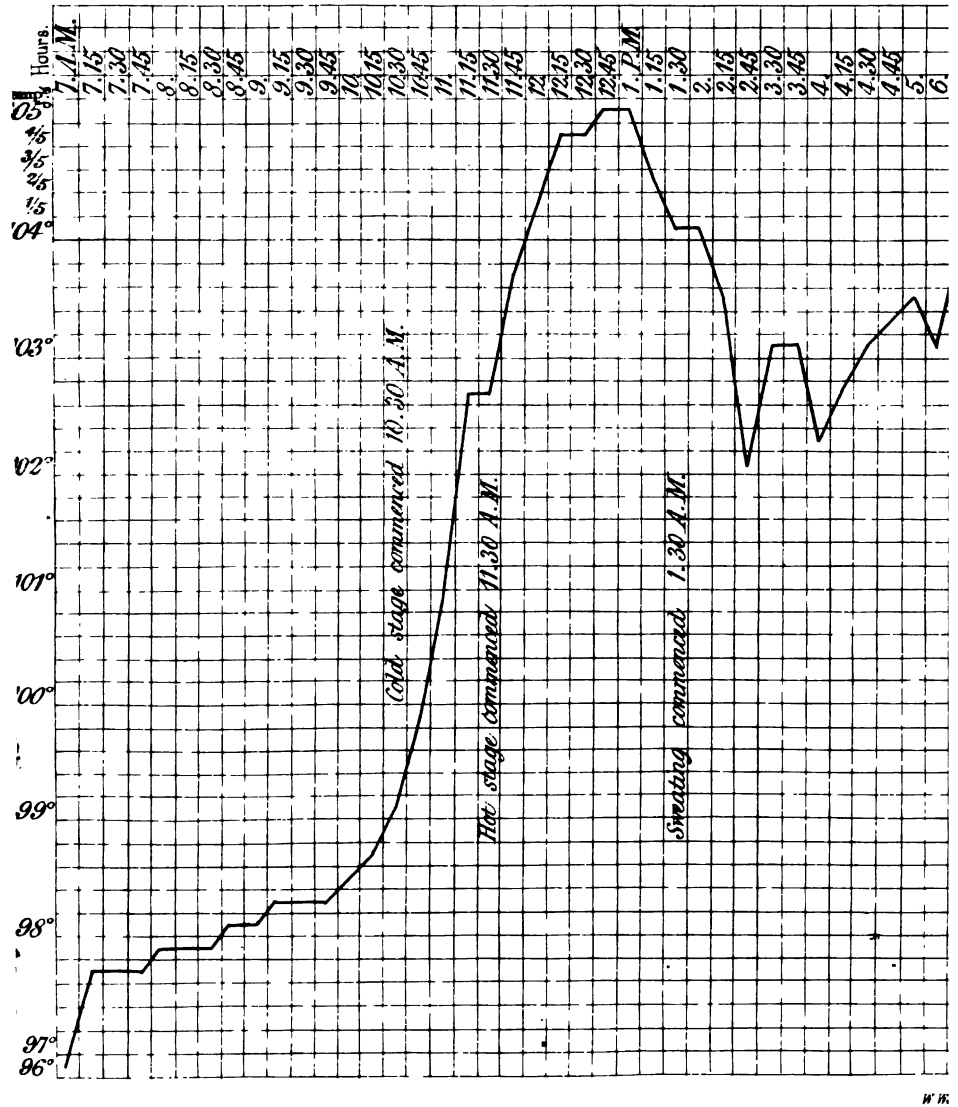
He passed the following quantity of urine:

Before fit (3 hours)	162 c.c.	= 54 c.c.	per hour.
Cold stage (1 hour)	100 „	= 100	„
Hot stage (2 hours)	180 „	= 90	„
Sweating stage (4 hours)	150 „	= 37½	„

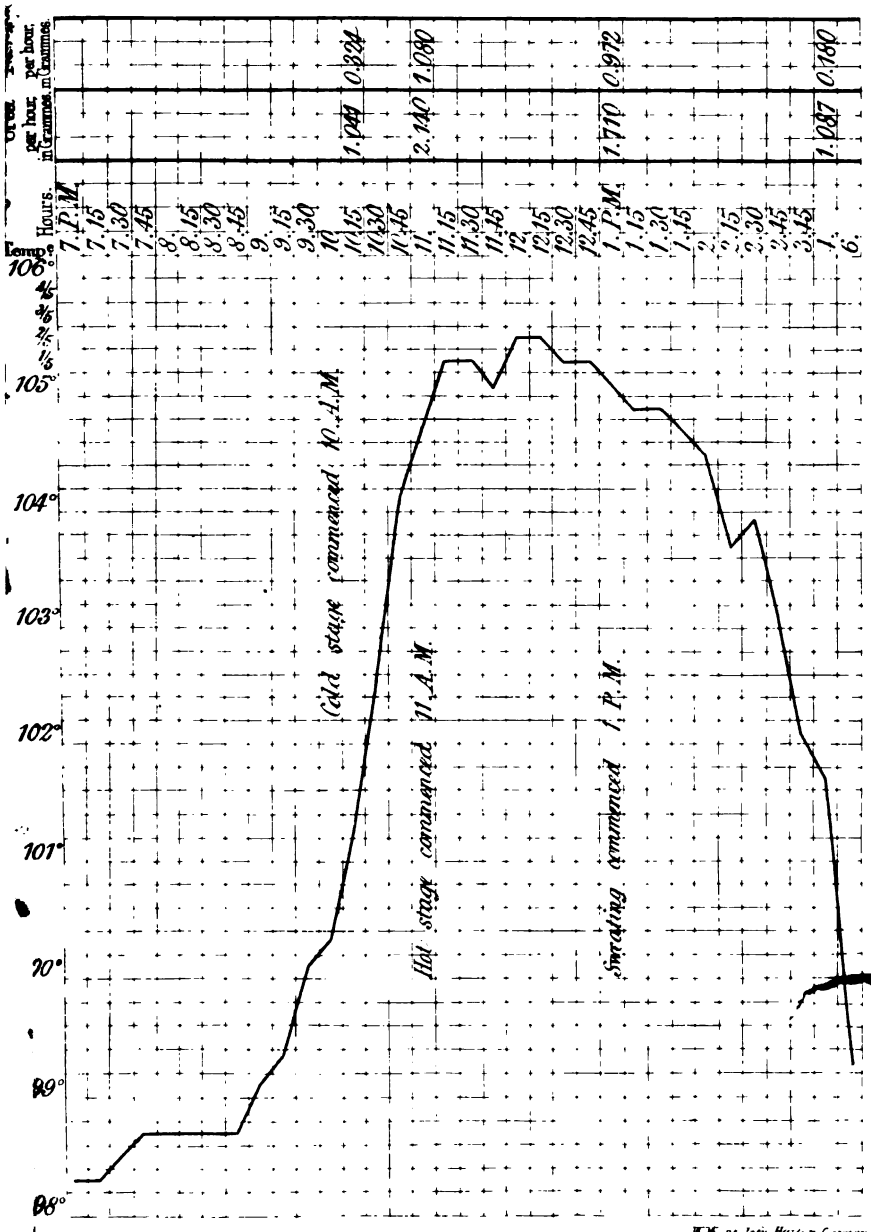
[1] The quantity of urea to each degree was 1·977 grm., showing a close correspondence to the amount for each degree in the former case. The correspondence would probably be closer still if the normal amount of this patient was reduced to that of the former.

[2] The amount of chloride of sodium corresponding to each degree was 0·502.

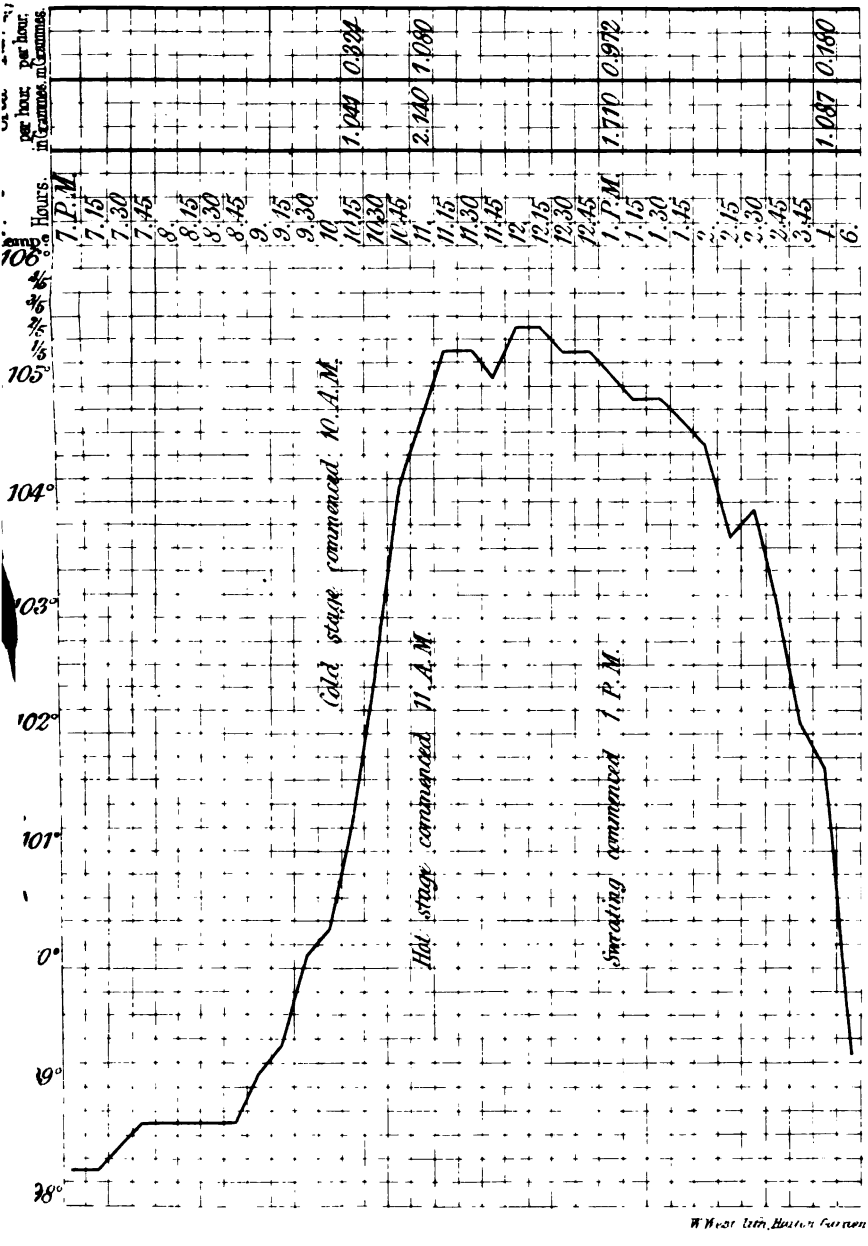
APRIL 2ND


Hours.
7 A.M.
7.15
7.30
7.45
8.
8.15
8.30
8.45
9.
9.15
9.30
9.45
10.
10.15
10.30
10.45
11.
11.15
11.30
11.45
12.
12.15
12.30
12.45
1 P.M.
1.15
1.30
2.
2.15
2.45
3.30
3.45
4.
4.15
4.30
4.45
5.
6.
105°
4/5
3/5
2/5
1/5
104°
103°
102°
101°
100°
99°
98°
97°
96°
Cold stage commenced 10.30 A.M.
Hot stage commenced 11.30 A.M.
Sweating commenced 1.30 A.M.

APRIL 4TH

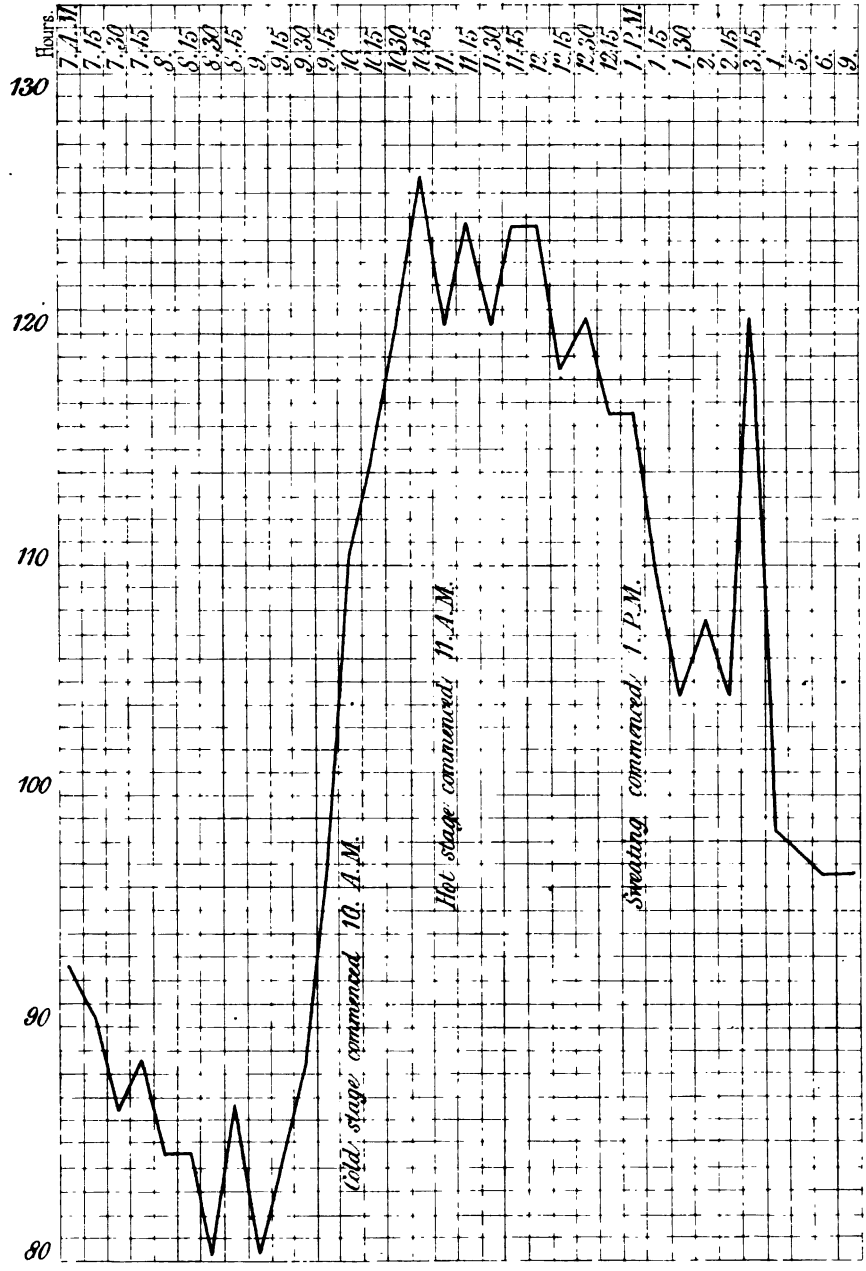

Oxygen per hour in Grammes
Carbonic Acid per hour in Grammes
1.044
0.824
2.140
1.090
1.710
0.972
1.087
0.180
Hours.
7 P.M.
7.15
7.30
7.45
8.
8.15
8.30
8.45
9.
9.15
9.30
10
10.15
10.30
10.45
11.
11.15
11.30
11.45
12
12.15
12.30
12.45
1 P.M.
1.15
1.30
1.45
2.
2.15
2.30
2.45
3.
3.15
6.
Temp.
106°
105°
104°
103°
102°
101°
100°
99°
98°
Cold stage commenced 10 A.M.
Hot stage commenced 11 A.M.
Sweating commenced 1 P.M.

APRIL 4TH


Hours.
7. P.M.
7.15
7.30
7.45
8.
8.15
8.30
8.45
9.
9.15
9.30
10.
10.15
10.30
10.45
11.
11.15
11.30
11.45
12.
12.15
12.30
12.45
1. P.M.
1.15
1.30
1.45
2.
2.15
2.30
2.45
3.
3.45
4.
6.
per hour in grammes.
1.044
2.140
1.710
1.087
per hour in grammes.
0.324
1.080
0.972
0.160
106°
105°
104°
103°
102°
101°
0°
99°
98°
Cold stage commenced 10 A.M.
Hot stage commenced 11 A.M.
Sweating commenced 1 P.M.

APRIL 4TH

Pulse.



Thus, during the period before the cold stage he drank 323 c.c. in excess over the quantity of urine passed.

Cold stage,	he drank	100 c.c.	more than he passed;
Hot stage,	„	520 „	„
Sweating,	„	350 „	„

making a total of 1293 c.c. in excess of the total amount of urine voided. In this respect, again, he corresponds to the previous patient.

The pulse was taken every quarter of an hour with the temperature in the two fits, and the result is shown in the two following charts. In the first the pulse at the very commencement falls somewhat, and then oscillates. Just before the commencement of the cold stage it commences rising, and then remains stationary for half an hour; then runs up enormously and rapidly during the whole of the cold stage, and at its termination remains again stationary for half an hour; then runs up slightly, oscillates when it has reached its highest point, and immediately on the sweating stage setting in it falls rapidly. In the second chart, at the very commencement, the temperature falls more than it did in the previous one; in the stage previous to the cold it rises more rapidly than in the previous one; runs up still more rapidly during the cold stage, reaches its highest point, and then immediately oscillating, before the sweating stage commences; after oscillating for some time, it falls, and at the commencement of the sweating stage it stands stationary for half an hour, and then falls greatly. During the entire rise there is no tendency for it to remain stationary, still less to oscillate.

This last fit was more severe than the former.

Thus, on both days the pulse commences by falling somewhat, then on both it rises before the fit commences, thus corresponding to the urine and its constituents and also to the temperature. At the commencement of the cold stage, in the less severe fit, the pulse remained stationary for half an hour, and again at the termination of the same stage, whilst nothing of this is seen in the severer one, thus cor-

responding to the temperature, &c. Again, on reaching the climax on both days the pulse oscillates, and the temperature also was unsteady at the same time, though to a much less extent. The pulse commences falling before the sweating stage, and then falls rapidly, more so than the temperature. The pulse, however, fell most rapidly in the less severe case.

Thus, a close correspondence exists between the pulse and the temperature. Like it, the character of the rise varies with the intensity of the fit; it rises before the cold stage commences; then, if the fit is not very severe, remains stationary; again rises through the entire cold stage, the rise being more considerable than during any other period; then falls before the sweating stage, and just at the commencement of this stage stands still for half an hour in the severer cases, and falls subsequently slower than in the less severe cases, in which also there is no stationary period.

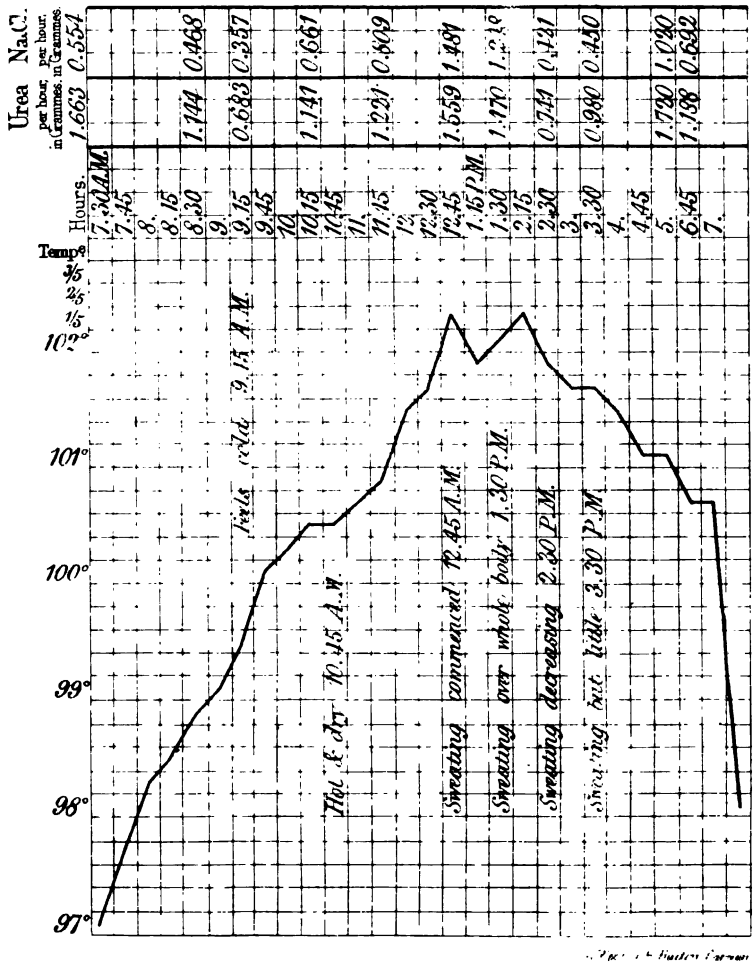
Case 3.—*A case of hectic fever.*—The following well-marked case of hectic is subjoined, on account of its close correspondence to the cases above given.

It occurred in a phthisical patient, æt. 45, under Dr. Walshe. It was particularly obstinate in resisting all measures for its removal, and was also peculiar in its occurring always during the day, and its very long continuance, as it commenced at 7 a.m., and continued to 5 p.m. All the stages were unusually well marked, consisting in severe rigors, accompanied with great pain about the loins and legs, the rigors coming on gradually, the sensation of cold being first observed about the feet; the hot and especially the sweating stages followed regularly. The observations were conducted in all respects after the manner of those already detailed.

Temperature.

Here, as in the other cases, the temperature began to rise before the rigors set in, ran up slowly through the

Case of Hectic.



whole of the cold stage, at its termination remained stationary for half an hour, then again ran up slowly, remaining stationary once, till it reached its highest point, when it oscillated, the rise in the oscillation occupying half an hour, then slowly and gradually fell, every now and then remaining stationary. A close connexion exists between this temperature and those given formerly; the rise, however, before the shivering set in was peculiarly great, and the cold stage very short, the rise in this stage not being so much in excess over the other stages as in the cases of ague.

Urea.

Unlike the cases of ague, the urea falls during the period preceding the rigors, and reaches its minimum immediately they set in; the quantity then rises rapidly, and becomes greatest during the hour immediately preceding the sweating stage. The quantity then falls rapidly through the sweating stage, reaching its minimum when the sweating is most severe; then rises again even higher than it was during the hot stage, and after that falls to its normal amount.

Chloride of sodium.

This corresponds to the urea in all respects, except that the rise at the termination of the fit does not equal the amount secreted during the hot stage.

Water

Again corresponds to both the above, except that its greatest quantity was poured out an hour later than the greatest amount of urea and chloride of sodium (showing the independence of these substances one of the other).

The patient was permitted to drink *ad libitum*; the quantity, however, was not measured.

The following table shows the variations in the amount of urinary water:

Hour.	Amount of Water.
7·30 a.m.	93 c.c.
8·30 „	52 „
9·15 „	35 „
10·15 „	58 „
11·15 „	71 „
12·30 p.m.	97 „
1·30 „	104 „
2·30 „	39 „
3·30 „	50 „
5 „	100 „
6·45 „	70 „

The pulse in this patient remained about 80 throughout the day.

General Conclusions.

The following are the conclusions deducible from the facts noted in the cases of ague.

Temperature.

1. This rises before the commencement of the subjective fit.
2. The time before the cold stage at which the rise commences varies.
3. It continues to rise during the entire cold stage.
4. The rise during this stage is greater than during any other.
5. It reaches its highest point during the hot stage, but falls again before the sweating stage, the fall being gradual.
6. The fall is more rapid during the sweating stage. The rapidity is in proportion to the slightness of the fit.
7. Definite variations occur in the rise and fall of the temperature indicative of alterations in the severity of the fit.
8. These variations first appear at the commencement and termination of the cold stage.
9. Their earliest indication is seen in a tendency for the later rises in the cold stage to become less extensive.

10. The temperature next becomes stationary for a variable time at either or both extremities of the cold stage.

11. Should the fit become still less severe, the temperature oscillates at these points.

12. An oscillation in the middle of the cold stage indicates a still further diminution in the severity of the fit.

13. Variations also occur during the hot stage.

14. In the severest fits there is a tendency for the temperature to remain stationary at its highest point.

15. If less severe, the temperature immediately falls on reaching its extreme height.

16. When the fits become still less severe the temperature next fails to reach to such a height as previously, but at this point again has a tendency to be stationary.

17. Should the severity of the fit be still less, it just touches the extreme point, and immediately falls.

18. Variations are also observed during the fall of the temperature.

19. Thus, at the junction of the hot and sweating stage, an oscillation, or a tendency to remain permanent, always occurs.

20. In continuing to fall, the temperature either falls gradually and continuously; this occurs in the severe fit—or,

21. Remains stationary every now and then; this indicates a somewhat less intensity of the fit—or,

22. It oscillates; this accompanies the most rapid fall, and occurs during the least severe fit.

23. After sinking to its extreme point, the temperature has a tendency to rise again, the rise being often considerable.

Urea, Chloride of Sodium, and Water.

24. The urea, chloride of sodium, and water, also begin to increase in quantity before the commencement of the cold stage.

25. They continue to rise rapidly, and become most

abundant either at the termination of the cold or the commencement of the hot stage.

26. These urinary constituents commence to fall in amount before the temperature reaches its highest point.

27. During the latter part of the hot stage they decrease in amount slowly.

28. They fall rapidly during the sweating stage, the rapidity of the fall being proportionate to the slightness of the fit.

29. These constituents exhibit variations corresponding to the variations in the temperature.

30. Thus, when the temperature remains stationary at either end of the cold stage, these urinary constituents also remain stationary, or fall somewhat. When the temperature oscillates, the fall in these constituents is proportionately greater.

31. A fall occurs in these constituents corresponding to the oscillation in the temperature at the termination of the hot stage, the fall in their amount corresponding to the depth of the oscillation.

32. A greater diminution in the amount of these constituents is observed when an oscillation occurs, than when the temperature decreases gradually, or tends to remain stationary.

33. The increase in the urea and water is definite, the same amount of increase corresponding to a single degree each day.

34. A greater increase in these constituents corresponds to a single degree at a high than at a low temperature.

35. No connexion existed between the various rises of the urea and chloride of sodium when equal periods were compared together.

36. The urea appeared to be independent entirely of the influence of the amount of water.

37. The chloride of sodium also underwent a definite increase, but this increased per-centage remained the same whatever the amount of water was, so that the total amount of chloride was greatly under the influence of the urinary water.

38. The chloride rose steadily and constantly, and did not observe the variations corresponding to temperature which were seen in the urea, but the water varying with the urea and influencing the chloride, as seen above, caused similar variations in the hourly amount of the chloride.

39. The quantity of water drunk in no way influenced the total amount excreted.

40. The increase in the above constituents often commenced before a corresponding rise in the temperature occurred.

41. The variations in the temperature above described often followed similar variations in the urea, but never preceded them.

42. Quinine given in a single, but large dose, when the temperature commenced to rise, lowered the temperature and postponed the fit for an hour, but had no other effect on that fit, though it prevented its recurrence next day, another scruple having been taken.

43. The pulse corresponded closely with the temperature.

CASE

OF

PARALYSIS

AS TO VOLUNTARY MOTOR POWER OF THE LIMBS ON ONE SIDE OF THE BODY,

ATTENDED BY

HYPERÆSTHESIA AS REGARDS THE IMPRESSIONS OF PINCHING AND PRICKING ON THE CORRESPONDING SIDE OF THE FACE;

BEING THE RESULT OF COMPRESSION OF CERTAIN LATERAL PARTS OF THE BRAIN FROM AN INTRA-CRANIAL ANEURISM:

WITH OBSERVATIONS

ON "INDUCED" CEREBRAL PARALYSIS.

BY

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ASSISTANT-PHYSICIAN TO ST. GEORGE'S HOSPITAL.

Received June 13th.—Read June 28th, 1859.

THE results of clinical research into diseased actions, and into their connexion with certain lesions found 'post mortem,' are so far uniform and constant that we have for some time had no difficulty in recognising the positive relation-

ship which obtains between certain affections of one side of the brain, and complete loss or impairment of motor power or sensation on the "opposite" side of the body, to be that of cause and effect, and not merely one of coincidence.

That this relationship might be expected to subsist we are, moreover, taught by the investigations of experimental physiologists into the natural anatomical disposition of various portions of the brain and spinal cord, and in no way more decisively than by the experiments of Dr. Brown-Séquard, recently brought before the notice of our profession in England.[1]

According to these delicate and complex experiments (observed with such apparent truthfulness, and exposed so luminously, that they must be regarded, I think, as exhaustive), all the will-conducting fibres passing down from the brain to the voluntary muscles of the body decussate each other at the anterior pyramids.

We are assured by that observer, that "if a section is made longitudinally just at the place of the decussation of the anterior pyramids, so as to divide entirely all the decussating elements, we find that, although the animal lives some time after the operation, it has no voluntary movement at all in any of its limbs, which are almost always the seat of convulsions. A section of the two anterior pyramids is followed by the same results, while a section of the olivary columns, which are chiefly the continuation of the anterior columns of the spinal cord, does not seem to produce a notable paralysis."[2]

Such relationship, then, appears to be the rule, as determined both by direct experimental research and by clinical observation; and so completely is it generally established, and so naturally is paralysis on a given side of the body expected from injury or disease of the opposite side of the

[1] 'Lectures on the Physiology and Pathology of the Central Nervous System,' delivered at the Royal College of Surgeons in May, 1858.

[2] See Lecture No. IV, as communicated to the 'Lancet' for July 31st, 1858, p. 111.

brain, that when apparent exceptions are encountered, the bystander is at once apt to conjecture that some error of observation has been committed. In some cases, it may be supposed that after death the entire brain has not been scrutinised with sufficient closeness in the search after morbid lesion; or that the examination of the various nerves has been overlooked; or that the spinal cord, of which some unsuspected affection may have coexisted as a complication, has, for some reason, or without any, been neglected.

But *bonâ fide* cases of exception to this rule are, without any doubt, duly authenticated, and we have instances on record, in which more or less interference with the power of movement or of sensation on one side of the body has been found, after death, to have been dependent upon organic lesion of parts within the cranium on the side corresponding to the paralysis, although our existing knowledge has been unable to afford any valid anatomical explanation of the connexion between the lesion and the symptoms.

In the exceptional cases to which I allude it has happened that the central injury or disease has not been at all restricted to any definite portion of the nervous system; but it is not a little remarkable that there is one portion of the brain, injury of or pressure upon which, if carried to a certain degree,[1] will *invariably* produce as it appears more or less complete paralysis of motor power on the "corresponding" side of the body (provided paralysis be at all produced), and that precise locality is the inferior surface of one of the crura cerebelli along with the commencement of the trigeminal nerve. This connexion has been abundantly substantiated by Dr. Brown-Séquard, who has collected and sedulously collated fourteen cases—which have, by various authors, been placed on record—strikingly illus-

[1] Should, however, neighbouring parts be considerably involved by the direct pressure of such a tumour, or should inflammation produced by its pressure extend at all deeply, we then have paralysis, not on the corresponding, but on the "opposite" side of the body, just indeed as if the affection of the particular part of the crus cerebelli had not primarily existed.

trating the statement. That observer[1] shows, that when a tumour exists between the inferior surface of the middle cerebellar peduncle and the petrous element of the temporal bone, so as to press upon the former without affecting the interior or the superior surface thereof, and at the same time to involve but slightly the neighbouring parts, we have an incomplete paralysis of motor power produced on the "corresponding" side of the body. This is attributable, as he suggests, to primary irritation or excitement either of the crus cerebelli, or of the dura mater, or of the root of the fifth pair of nerves; but most probably it is due to irritation of the anterior (or inferior) part of the cerebellar peduncle.[2]

In the concluding part of the twelfth lecture above alluded to, a woodcut (fig. 25) is given by Dr. Brown-Séquard of the base of the brain, showing the anatomical relation of the various parts above mentioned, pressure of which by a tumour (at the point marked *c c*) is said by him almost invariably to produce this form of paralysis.

Singularly enough, the case of which I am about to give the details (and the records of which have for some time rested in my note-book, along with the accompanying illustration, Plate VI., uninterpreted, and therefore unused, until rendered significant by reading the lecture above mentioned), exemplifies, to a nicety, pressure upon the exact spot indicated in the fore-named woodcut, by means of an aneurismal tumour, which also implicated the apparent root or central connexion of the fifth cranial nerve, as well as the

[1] Lectures in the 'Lancet,' Dec. 25th, 1858; also Brown-Séquard's 'Journal de la Physiologie de l'Homme et des Animaux,' vol. i, p. 534.

[2] Dr. Brown-Séquard grounds this view mainly on the fact that the cerebellum into which the peduncle passes is itself capable, under diseased action (although it does not always do so), of producing a similar form of paralysis (see 'Journal,' sup. cit., p. 535). The similar form of paralysis alluded to is that termed amaurosis, which cannot, of course, be an "immediate" one, inasmuch as the cerebellum is neither a centre of volition or sensation, nor yet a channel for the conduction of sensory or motor fibres.

inferior surface of the middle cerebellar peduncle on the left side. This case, which I will at once proceed to relate, was as follows:

Epilepsy; complete loss of sight for five years; impairment of the senses of smell and taste on the "left" side; partial loss of muscular power on the "left" side of the body; contactile hyperæsthesia of the skin of the "left" side of the face and head; aneurism of the anterior cerebellar artery on the "left" side, compressing the "left" crus cerebelli, and the contiguous portions, to a slight degree, of the pons Varolii, cerebellum, and apparent root of the fifth nerve.

Previous history.—The patient, Mrs. S—, æt. 46, and the mother of several delicate children, came under my care in December, 1851. She had always enjoyed tolerably good health until the year 1848, when she began to complain of pain at the front and vertex of the head, along with dimness of sight, which was especially worse on stooping, and pain referred to the back of the eyes. Occasionally she would entirely lose her sight for a few seconds at a time, the defect of vision "beginning at the upper part of the eyes," to quote the patient's own words, the upper part of any object regarded becoming invisible, whilst the lower part was still seen. I could not establish the fact that sight had failed more completely or sooner in one eye than in the other. She was also subject to feelings of stupidity and heaviness, which increased from the first. Shortly before I saw her she had had several epileptic seizures, during which she bit her tongue, which lasted about ten minutes, and followed an attack of giddiness; and it was for one of these attacks that she came under my observation. About one year after the commencement of her illness, she became totally blind of both eyes, and was placed under the charge of one or two oculists.

Symptoms when first seen.—At this time (December,

1851), I found her pale in the face, but she was, however, pleasing in countenance; the mouth was drawn to the right side, the left side of the face and forehead generally being, comparatively speaking, deficient in wrinkles and power of expression. The pupils of both eyes were very large, but equal in size, and with a regular outline, and scarcely at all acted under the influence of light. There was no ptosis of either upper eyelid. The patient complained of great deafness in the "left" ear, but had never suffered from any discharge from the ears. The tongue was protruded in a straight direction. The patient had a want of power in properly blowing the nose, and seemed almost entirely to have lost the sense of smell on the "left" side of the nose; that of taste appearing quite unaffected. As to her mental faculties, she was generally very intelligent and spoke and answered questions fluently and sensibly. At times, however, her memory was deficient, and this was so especially as regarded recent events, whilst most present subjects appeared to the patient, as she said, "far off in thought." Her husband observed that she was wont to understand what was going on at the time, but would also talk of circumstances long past as if they had but just occurred. There was a loss of power, although incomplete, down the whole of the "left" side of the body, and also great *increase of sensibility of the skin covering the left side of the face and head.* This hyperæsthesia of the scalp was constant, and so great that the mere *touch* or pressure of her ordinary bonnet was most painful to her. Pain also existed in the back, but none was complained of in the abdomen. The urine was at times voided with difficulty, and there was an abundant dark-red vaginal discharge. Occasional palpitation of the heart also existed, but the sounds of the heart and lungs were natural; the pulse was regular, although quicker than it should be. The gums and tongue were rather affected with mercury, which she had been taking.

When I next saw her, which was not until March in the following year, she expressed herself as being "better in

health." There was the same distorted condition of the features, the mouth being, as before, drawn to the right side, and the eyes still blind; but she said that she had a "sort of a flash" in the right eye at times, the pupil of which was now larger than that of the left one. There was also a feeling as if something unusual existed behind the eyes, especially the left one. At times, she had pain in the forehead. The memory and senses of smell and hearing were as deficient as before, and a swelling of the right side of the head and neck had come on, which was very tender to the touch, and was at times the seat of a sharp pain darting to the right ear, and giving the sensation of pressure being exercised upon it from within. At times, also, she had a troublesome "twitching of the left arm," which became painful when laid upon in bed; but the limbs on the left side had recovered some of their power, and the increased sensibility of the integuments of the head and face on this side had to a certain extent diminished. The pulse was pretty good; the functions of the stomach, bowels, and liver were active.

In the month of April the pain in the head and neck was much less; but she complained of pain, with a feeling of numbness and stiffness, in all the limbs, and she was often overcome by drowsiness. There was, however, no proper anæsthesia as regards the impressions of pricking, pinching, &c., in any part of the skin. She complained of frequently having very odd sensations, fancying that she had water in the head, which caused pain in lying down; and she frequently thought that she heard the noise of a "sawing" in the head on any excitement. She was also subject to a feeling of sickness in a morning, but this had not amounted to actual vomiting.

In May, I was sent for to see her at Blackwall, and found her not so well, as she appeared to be debilitated by the use of remedies resorted to. In September she still retained the odd feelings before complained of, and frequently had "twitchings" and "contractions" of the muscles at the right side of the neck and shoulders, and of stiff-

ness of the left side of the neck and lower jaw. Anæsthesia, as to pricking, pinching, &c., was wholly absent; but now the sense of taste had become interfered with on the "left" side of the tongue, the power of swallowing remaining good. She told me at this time of her sons having become the subjects of consumption.

After the above date, I never again saw the patient alive. She appears to have gone on much in the same condition for about two years, during which time she had occasional returns of convulsive attacks, but scarcely to have suffered in any way from headache. At the end of the above period she heard of the death of one of her sons, and immediately had a violent epileptic seizure, for which she was attended by Mr. Bain, of Poplar. In a letter, which that gentleman kindly sent to me, he states that for several months she enjoyed exemption from the fits, but in September, 1856, she had one of a very aggravated character, the right side of the body being tolerably quiet, whilst the "left" arm was much convulsed, and the "left" eyelids continually winking. The right eye was, however, kept open, and the pupil of both eyes dilated, both eyeballs rolling about from side to side. The patient died immediately after this violent attack.

Treatment.—As I have before stated, when I first saw this patient she was under the influence of mercury, which, along with small quantities of ipecacuanha, and occasional aloetic purgatives, had been resorted to, under the impression that she was the subject of some form of intra-cranial chronic inflammation. Agreeing in this assumption for some time, I determined to continue this method of treatment, but with caution, combining with it the use of good nourishment. Afterwards, however, especially when I learnt her family history, and found that she became weaker, I discontinued the mercury, and gave her quinine and strychnia, which evidently agreed with her. Under their use, according to her husband, she became freer from pain, her appetite was improved, and her general comfort

and happiness increased. The convulsive attacks appeared to be diminished in duration and number from the use of a seton in the neck, which, towards the close of her life, Mr. Bain judiciously persuaded her to submit to.

Post-mortem examination.—On examining the cranium and its contents, along with Messrs. Bain, sen. and jun., the following condition of things was found. The blood-vessels of the scalp were very congested. The bones of the skull, as also the dura mater and the arachnoïd membranes generally, were natural, but here and there the latter membrane was a little thickened at the summits of the cerebral hemispheres. A large amount of clear fluid existed in the ventricles of the brain and subarachnoïdean spaces, and the superficial vessels of the brain were very full of blood. The brain-substance was healthy to the naked eye, but the large and smaller superficial arteries at the base of the brain were highly atheromatous.

Connected with the anterior cerebellar artery on the "left" side of the brain was an aneurism of about the size of a small nutmeg, resting immediately upon the inferior surface of the left middle crus cerebelli, and indenting, although very slightly indeed, the contiguous structures of the pons Varolii and left lobe of the cerebellum (see Plate VI), which parts at the point of contact were very superficially softened. Moreover, the apparent root of the fifth or trifacial nerve (see Plate VI, fig. 3) was pressed upon by the aneurism anteriorly; and the seventh nerve, on the same side, as it passed forwards at the base of the cranium, was greatly implicated and stretched by it. In shape the aneurism was irregular and somewhat nodulated, its surface being very firmly adherent to the corresponding part of the dura mater at the base of the brain, and, by reason of the rupture of its adhesions, necessitated by the removal of the organ, very roughened. When cut into, it was found to be quite solid and full of firm material, part being altered fibrin of a yellow and pale-red colour, and part being composed of dark blood-clot. It was surrounded by a dis-

tinct resisting but thin membrane, evidently the thickened coat of the blood-vessel, which could be without difficulty dissected off.[1]

The optic nerves, commissure, and tracts were very dwindled and softened, and of a semi-transparent yellowish colour, as also was the seventh nerve on the left side, in the neighbourhood of the aneurism.

Microscopical appearances.—On minute examination the atrophied and discoloured optic nerves and tracts and seventh nerve were seen to present, in addition to a few broken remnants of nerve-fibres, a large amount of granular matter and many delicately contoured round and oval-shaped nuclei of small size, with, here and there, a considerable quantity of fatty material.

No proper or healthy nerve-structure was found in these parts. The roughened surface of the aneurism, where adhesions to the dura mater had occurred, presented structures which are commonly expected among rather newly formed adhesions, such as delicate fibres, and also granular and slight fatty matter, with occasional blood-vessels possessing very delicate walls, containing spare elongated nuclei. In some places a few so-called granular corpuscles of large size existed.

In various parts of the brain small capillary vessels were met with in a state of "fatty degeneration," but beyond these particulars nothing unusual was found in connexion with the brain.

Remarks.—Respective of the diagnosis during life in this case, it proved that I was totally in error as regards the exact cause of the symptoms.

I was in no ways prepared to discover an aneurism to be the means of compression within the cranium; but that pressure, from some cause, was being exercised, and that at the base of the brain, I felt quite assured; and I fully

[1] The preparation showing this aneurism of the cerebellar artery will be placed in the St. George's Hospital Pathological Museum, as No. 3, *b*, Sub-series ii, Appendix to series xxi.

anticipated that we should meet with a scrofulous tumour within the cranium. The nature of the pain, the absence of any great mental disturbance, the blindness, and the existence of facial paralysis and deafness on one side, pointed to the base of the brain as the locality where pressure was being exercised. From the history and appearance of the patient, as well as from the fact that her family were very delicate, and that of her children two had died of phthisis, I was eventually possessed by the opinion that the supposed growth or tumour was, in all probability, one of a scrofulous nature. As regards the various symptoms and their value, the most notable was doubtless the fact of the incomplete loss of power of motion in the arm and leg on the "left" side, that, viz., corresponding with the brain-lesion, and this, too, without true anæsthesia in any part of the body; whilst at the same time there was greatly exalted sensibility of the skin of the face and head on the same side. The dependence of these symptoms upon such a lesion is certainly quite anomalous, and a few words may well be spent on their consideration in reference to that lesion.

In the first place, the character of the symptoms showed that the lesion was, at any rate, a cranial, and not a spinal one; and, as before said, that it was situated somewhere at the lower part of the encephalon. Could the lesion have been considered during life as one of the pons Varolii or of the medulla oblongata, or of the crura cerebri or cerebral hemispheres, &c.?

The admirable and extensive researches of Gübler and Brown-Séquard demonstrate that if a lateral part of the pons Varolii be so greatly affected as to lead to paralysis of motion in the limbs, that paralysis is on the side of the body "opposite" to that on which the lesion exists; and Gübler has pointed out that if facial paralysis be produced in such cases, it is generally "*not*" on the same side as that on which the limbs are affected, but on the side on which the encephalic lesion exists.

Moreover, in injury of this exact portion of the nervous

system, we have produced, as the ordinary rule, destruction or impairment of cutaneous sensibility on the side "*opposite*" to the lesion.

Considering these facts, the intra-cranial lesion in the present instance could not have been looked upon as existing in the pons Varolii, inasmuch as there was altogether an absence of anæsthesia; and, moreover, the facial paralysis was on the same side of the body as the diminished power of moving the limbs; whereas, where it exists along with more or less hemiplegia in connexion with injury or disease of the pons Varolii, it is most frequently observed on the side of the face opposite to that of the paralysis of the limbs. Still less was there evidence of lesion of the central part of the pons Varolii, for under such circumstances we should have had produced loss or diminution of power in the limbs of "*both*" sides.

Nor, again, were the symptoms suitable to lesion of the medulla oblongata, for then we should have had no facial paralysis, unless the cause of pressure had so extended in an upward direction as, in addition to the injury to the medulla oblongata, to affect the junction of the seventh nerve with the pons Varolii. Moreover, in this case, as in the instance of lesion of the pons Varolii, we should, as a rule, have had some degree of cutaneous anæsthesia on the same side as the paralysis of the limbs.

Neither, again, could the symptoms have been considered as tallying with lesion of the cerebral lobes, of the corpus striatum, optic thalamus, or crura cerebri, &c.; for in disease of all of these parts we should expect impaired cutaneous sensibility on the same side of the body as that on which impaired motor power existed.[1]

[1] In connexion with this case I have been unable to say anything as regards the temperature of the skin (a subject of such interest as regards the diagnosis of disease or injury of the brain or spinal cord), inasmuch as no alterations in this respect were noticed during life. Nor, indeed, if they had been remarked, is it probable that in so long continued a case they would have been so invariable as to have afforded any reliable data for useful inference.

Seeing then that the symptoms, although indicative of disease of some parts of the base of the brain, did not harmonise with lesion of any of the above-mentioned parts of the encephalon, and also considering that the third, fourth, and sixth cranial nerves were unimplicated (as shown by the complete absence of ptosis or any interference with the movements of the eye-balls), it only remained that some part of the cerebellum or of its crura should have been considered as affected. But inasmuch as the two divisions of the seventh nerve on the "left" side were interfered with in some part of their course (as was obvious from the deafness and the paralysis of the facial muscles), it would seem that the exact point of lesion could not be very far removed from the central line, and therefore (apart from other reasons) it was highly improbable that the cerebellum itself would be its seat.

By the method, therefore, of exclusion or isolation, one would be compelled to locate the lesion in one of the crura cerebelli, and thus in the present case, as well as in the other like fourteen cases to which I have before alluded as having been placed on record, it becomes necessary to establish a relation of cause and effect between the presence of morbid phenomena as regards muscular action in certain parts of the body, and lesions of such portions of the nervous centres as are not known (as far as hitherto has been gathered from the results of vivisection) to possess, in a state of health, any specific influence upon those parts.

It may then be asked,—What explanation can be assigned of the existence of paralysis (more or less complete) of the muscles of the limbs as resulting from lesion of the middle crus cerebelli; and by what tract or nervous channel in such an anomalous case as the present one, has the morbid impression (whatever that may be), caused by the aneurism, been carried *to all appearance* vertically downwards from one side of the brain to the muscles on the corresponding side of the body?

Of course the answer to this question must, in the absence of any anatomical facts supporting it, be purely

hypothetical; but I see no reason why we should not (in the present state of our knowledge) accept the explanation put forth by Dr. Brown-Séquard—that in such a case as the one which I have just related, the paralysis ought to be regarded as similar to what has been termed the "reflex" paralysis due to an irritation of centripetal nerve-fibres in any viscus, any membrane, or the trunk of a nerve; and that it is not, indeed, the result of any "absence of action," or impossibility from some cause or other, of, or obstruction to, nervous transmission along certain voluntary nerve-fibres in the immediate neighbourhood of the lesion; but, on the contrary, that it is the result of an "excess of action" or irritation, exercised on some particular part, and acting in a direction from the affected region towards and upon some other part either central or conducting, so as (by some disturbance of nutrition, it may be) to bring about a paralysis or cessation of action in the nerve-fibres connected with that other supposed part; just as if that other supposed part had been primarily affected by some cause producing "absence" of its action.

That in such a case as the present one, the phenomena may arise from any implication of the small number of motor nerve-fibres which at a former period were conjectured by physiologists to exist as passing down (according to an ordinary arrangement) in a vertical direction from one side of the brain to the corresponding side of the spinal cord, thus avoiding the interlacement or decussation to which all the other motor fibres in the medulla oblongata are subject, cannot be supposed.

The knowledge which we now possess, attained chiefly by direct experiment, concerning the exact course taken by will-conducting fibres, or at least by volitional nerve-influence, renders such a view completely untenable. But even were motor fibres known to exist as thus in their course escaping the ordinary inter-crossing at the anterior pyramids, still their presence could not be considered as explaining these enigmatical phenomena, because in such a case as the present we have lesion of a part with which volitional nerve-

fibres on their course downwards towards the medulla oblongata have ordinarily no possible connexion.

Can we explain the subject under the supposition that in such a case as the present we have an instance of motor nerve-fibres, or nerve-influence, taking an unwonted or fortuitous course, finding their way in an abnormal manner from the cerebral hemispheres into the crura cerebelli, and getting down to the spinal cord eventually by the restiform bodies, so escaping any decussation?[1] Even allowing the possibility of irregularity of distribution of intra-cerebral nerve-fibres, it can, I think, hardly be maintained as

[1] Of course there can be no abstract difficulty in surmising that in some individual adult persons we may have an irregularity of distribution of nerve-fibres as well within as external to the great nervous centres. This general supposition receives support from the fact, that the intermingling or decussation of nerve-fibres which exists in the composition of the optic commissure is sometimes found in man to be defective, or even altogether wanting, as exists naturally in some of the lower animals. An instance of this observation in the human adult is mentioned by Vesalius, in a section in his anatomical work concerning the Optic Nerve (see vol. i, p. 366, of Boerrhaave's edition); and a small woodcut, showing the total absence of any chiasma, accompanies the description. Another instance is related in the Catalogue of the Museum of the late Mr. Langstaff (see Preparation No. 655). This specimen was purchased by Mr. Hillman, and presented to the Westminster Hospital; but having it removed from the bottle, and carefully examining it, with the help of that gentleman and Mr. Christopher Heath, it was found that the optic commissure really does exist, although, along with the optic nerves and tracts, it is in a very dwindled state. The subject of mal-distribution of nerve-fibres within the nervous centres is also illustrated by cases of deficiency of commissures, as of the corpus callosum, and by those cases (called by the French Diastématomyélie) in which, owing to some influence producing arrest, retardation, or perversion of growth, a bifid condition of the human spinal cord remains; a non-union remaining of the two juxtaposed longitudinal columns, of which, at one period in the course of development, the cord consisted. Such conditions of the spinal cord are alluded to by Andral, Ollivier, Vrolik, and many writers on Teratology; and generally occur in cases of acephalia and fissure of the bony spinal column. The subject is, moreover, exemplified by the fact stated by comparative anatomists, that in fishes the fibres of the spinal cord do not decussate.

affording an explanation of such cases as the one under consideration, inasmuch as it would not be at all probable that such an irregularity or misplacement of nerve-fibres would exist in almost every case in which a particular part of a particular crus cerebelli is injured in a particular manner.

We must then, I think, adopting an alternative course, accept the hypothesis suggested by Dr. Brown-Séquard as explanatory of those cases in which paralysis of the muscles on one side of the body is brought about by lesion on the corresponding side of the brain, and with him assume that the paralysis is one of an irritative character, and produced by an "*excess*" of action.[1] On this form of paralysis Dr. Brown-Séquard has lately to a considerable extent developed his views, whilst considering[2] the functions of the pons Varolii with reference to its various conducting and dynamic properties. In so doing it will be seen that he has given to this form of paralysis the distinguishing epithet "reflected" or "sympathetic;" or, again, "active" or "mediate," placing it in opposition to that other and more usual form which he terms "passive" or "immediate," and which is evidently the result of pressure immediately applied to nerve-fibres in direct communication with the muscles paralysed.

But under correction of so astute an authority, whilst fully and implicitly allowing the general method of explanation suggested by him to be the most probable one, I would venture to designate this kind of paralysis "induced cerebral" paralysis, thus bestowing upon it a title less likely than "sympathetic" to create confusion between this variety of paralysis, and that other of which, having its origin in

[1] The frequent existence of twitching or contraction of the affected limbs accords well with the supposed irritative character of the cause of the paralysis.

[2] See the valuable papers in his 'Journal de la Physiologie,' entitled "Recherches sur la Physiologie et la Pathologie de la Protubérance annulaire," Nos. 3 and 4, 1858, pp. 523 and 755; and No. 1, 1859, p. 121.

irritation of thoracic or abdominal viscera, or distant surfaces, we know the true sympathetic or ganglionic (so-called) system to be the medium.

The adjective "induced" is, I think, an especially advantageous one, as being more significant and expressive of the nature of the process through which this form of paralysis is supposed to be effected; an action so wholly different from, and indeed opposed to, that which causes the diagonal form of paralysis ordinarily met with. For whereas the term "reflected" would merely imply that the specific kind of action caused by a given lesion being transferred to some central part in another portion of the nervous system is simply reflected or diverted, and by means of efferent nerve-fibres operates on certain distal parts of the frame, we must on the other hand suppose that in these cases of paralysis which I would denominate "induced" the irritative action is carried, by the intervention of commissural fibres, from a given point to another part (and that too of the "opposite" side) of the brain, and there induces a repressive or inhibitory action of some kind or other[1] (but at any rate one wholly different in quality to that which was originally started) of motor nerve-fibres.

Thus, in short, we have an irritative action starting from the point of lesion, and so operating as in some manner or other to paralyse certain motor fibres in the opposite side of the brain, which, decussating at the anterior pyramids, affect the muscles on the side of the body corresponding with the original encephalic lesion.

It remains to consider one or two other symptoms in the case, which, although of minor importance compared with the hemiplegic symptoms, are worthy of special observation.

For instance, the partial impairment of the sense of taste which came on at a late period in the history of the disease, was of course referrible to paralysis of the lingual or

[1] Dr. Brown-Séquard is inclined to look upon this secondary action as producing a disturbance of nutrition upon which the paralysis depends. See the Journal above cited, vol. i, p. 536.

gustatory branch of the third division of the fifth cranial nerve;[1] but the complete integrity of the power of protruding the tongue and of deglutition, and the absence of noticeable interference with the functions of the heart, lungs, and larynx, &c., indicated that the ninth and remaining cranial nerves were uninjured.

Again, the hyperæsthesia of the skin of the face and head on the side of the body corresponding to the paralysis of the limbs was a symptom pre-eminently deserving of regard. This increase of sensibility of the part (so intense that acute pain was occasioned by the mere contact of the bonnet) must doubtless be referred to irritation or excitation of the apparent commencement of that most sensitive of all the nerves of the body, the fifth cranial nerve; and a glance at the accompanying illustration (see Plate VI, fig. 3) will show how this nerve at its point of union with the central nervous mass was pressed upon by the aneurismal tumour.

The subsequent decrease of the exalted sensibility of the skin of the face and head, as well as the partial re-acquirement of muscular power in the affected limbs, were probably due to diminution of pressure, owing to some gradual lessening of the aneurismal tumour in consequence of desiccation and shrinking of its contents.

To irritation of the motor or smaller division of the fifth cranial nerve must also be attributed the stiffness of the lower jaw (the temporal and pterygoïd and masseteric muscles being affected).

It is worthy of notice that the nutrition or vascularity of the eyeball on the side affected did not appear to be disturbed.

The attacks of epilepsy to which the patient was subject form also a salient feature in the history of the case, and point decidedly to irritation of some part, it may be of the fifth cranial or some neighbouring nerve, or it may be of

[1] The fact that loss of taste was not complete, whilst the fifth nerve was so considerably affected, appears to corroborate the proof, derived from other sources, that the glosso-pharyngeal nerve (in this case uninjured) is associated naturally with the fifth nerve in the function of taste.

some part of the cerebral membranes to which the aneurismal tumour had become adherent, and to which, as it is well known, so much nerve-influence from various sources is derived. To the frequent epileptic seizures no doubt it was that the defect in the patient's memory was due, as also the peculiar subjective sensations of which she latterly complained.

Finally, the loss of vision is not without interest.

Dr. Brown-Séquard shows that injury of one of the tubercula quadragemina produces a "*crossed*" paralysis of sight, whilst lesion of the pons Varolii, of the cerebellum or its peduncles, causes loss of vision on the side "*corresponding*" to the brain lesion. Can it be that injury to a single crus cerebelli, or to the cerebellum, is occasionally capable of affecting sight in "both eyes," and that simultaneously? The facts of the present case, taken in conjunction with the *à priori* reasonableness of the supposition, as afforded by the peculiar anatomical arrangement of the nerve-fibres at the optic commissure, lend some colour to the hypothesis.

The peculiarity which existed in this case as regards the power enjoyed by the eyes of only seeing certain parts of an object at a time, was no doubt attributable to a partial implication only of the nervous or vascular elements of the retina, in the first instance.

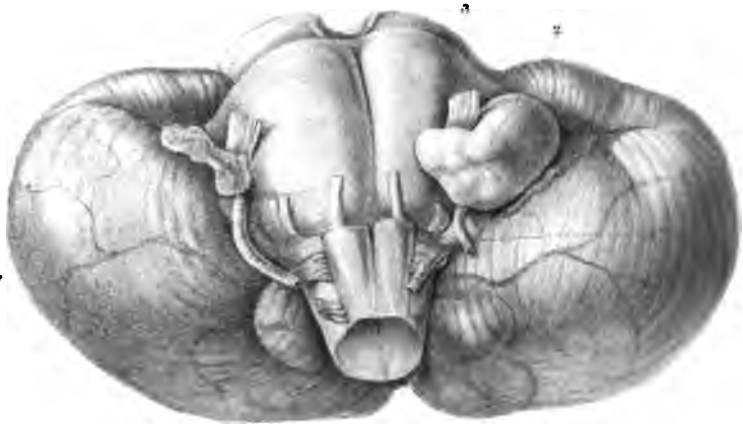
To the above case of paralysis I might have added others of which I have notes, in whose history paralysis of motion, with or without anæsthesia, was noticed as consequent upon disease or injury of the "corresponding" side of the brain.

This case I have selected, partly as being one of a comparatively rare nature as regards the relation between the aneurism and the particular part of the brain affected by it, and partly as being especially adapted, by reason of its simplicity and the freedom from complicating phenomena, to illustrate the general subject of what I have ventured to term "induced" cerebral paralysis.

DESCRIPTION OF PLATE VI.

The drawing represents an aneurism of the anterior cerebellar artery on the left side resting upon the middle crus cerebelli, the contiguous parts of the pons Varolii and cerebellum, and the apparent origin of the fifth or trigeminal nerve.

Fig. 1.—The anterior cerebellar artery.
„ 2.—The aneurism.
„ 3.—The root of the fifth nerve.



J H C del G.H.Ford lith W.West imp.

THE SEQUEL OF A CASE

(PUBLISHED IN VOLUME XLI OF THE TRANSACTIONS OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY)

OF

LITHOTRITY,

IN WHICH A COMMUNICATION EXISTED BETWEEN THE BLADDER AND INTESTINE.

BY

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Received June 27th.—Read June 28th, 1859.

In the last volume of the 'Transactions' of the Society there is an account of a case, in which a communication existed between the bladder and intestine, where a calculus had formed in the bladder, which I removed by lithotrity.

The patient having since died, I have thought that the following account of what was found at a post-mortem examination, might prove of sufficient interest to occupy the attention of the Society for a short time.

On February 2d, 1858, the patient was reported to be quite free from calculus in the bladder; and there were no symptoms of stone from this time to that of his death, which took place on April 19th, 1859.

He continued to pass fæcal matter occasionally with his

urine, and until within a few weeks of his death he daily washed out his bladder with warm water, by means of a syringe and catheter. About three or four months previous to his death his general health gave way; his digestive organs became impaired, accompanied with considerable irritability, and some mental disturbance; the symptoms exhibited previous to his death did not appear to depend upon the disease of the bladder or intestine.

Mr. Shield, of Hungerford, under whose care he was latterly, examined the body after death, and was good enough to send me an account of what he found, and also the bladder, and the portion of the intestine implicated in the disease, which are now in St. George's Hospital Museum. The following are the appearances they presented.

There was an opening in the bladder at the lower part of the posterior wall, of the diameter of a goose-quill, evidently not of recent date; the bladder corresponding to this aperture was intimately united by old adhesions to that part of the circumference of the sigmoid flexure of the colon that lies nearest to it. The aperture in the bladder communicated with the sigmoid flexure opposite their point of union. Above the point of communication of these two viscera, for the extent of about an inch, the canal of the sigmoid flexure was somewhat constricted; but this constriction was apparently due to the adhesion and subsequent contraction of these viscera, as beyond the point where the adhesion between them existed, the calibre of the sigmoid flexure appeared normal. Below the communication between the bladder and colon, the canal of the intestine was greatly constricted, to the extent of an inch and a half in length, admitting a tube through it of the size of the little finger. This stricture appeared to depend upon great condensation and subsequent cicatrization of the submucous and muscular tissues at that point. The mucous membrane of the intestine, above the seat of stricture, presented in many places pouches, varying in size from that of a pea to that of a filbert, and formed by protrusion of this coat externally. Opposite to the stricture it appeared

to be healthy, but very densely convoluted. The rectum was very much dilated, and had, during life, evidently acted as a second bladder, as from the symptoms described by the patient, the urine used to accumulate there, and was discharged in considerable quantities per anum. The bladder was healthy, and did not contain any calculous matter. The kidneys were somewhat congested, but otherwise were in a normal condition.

The appearances described bear out the opinion I gave in my former paper.

The history of this case is a good example of the great value of lithotrity in the treatment of calculus in the bladder in complicated cases. I was enabled, by this operation, entirely to remove the stone, and relieve the patient of great suffering; and I think, considering the state of parts revealed by the post-mortem examination, that, had lithotomy been resorted to, it would not have been followed by the same amount of success.

Mr. Sydney Jones, in December last, exhibited at the Pathological Society a specimen very similar to that I have described, and where a calculus was formed in the bladder, with fæcal matter as a nucleus; but no attempts appear to have been made to remove it, extravasation being the immediate cause of death. Mr. Jones observes—"The cause of death was the existence of calculus in the bladder. Had not this impediment to the escape of urine from the bladder been caused by the presence of this calculus, it is probable that the case would not have had so speedy a termination."

A CASE

OF

VESICAL CALCULUS

OF UNUSUAL SIZE

REMOVED BY THE RECTO-VESICAL OPERATION.

BY

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Received June 27th.—Read June 28th, 1859.

THE recto-vesical operation, though frequently performed by Continental surgeons for the removal of vesical calculi, appears to have been but little resorted to in this country, the lateral method having proved so successful in the hands of British surgeons as to have led to its almost universal adoption in cases where lithotomy is deemed advisable. But cases of a complicated nature occasionally present themselves, which justify departure from the ordinary rule of practice. A remarkable instance of this kind, I believe, recently came under my own notice, in which, for reasons to be hereafter stated, I considered the recto-vesical operation especially applicable.

James H—, æt. 21, was admitted into the Manchester Royal Infirmary, September, 1858, under the care of Dr.

Roberts, labouring under great irritation of the bladder. He was forthwith sounded, and a large stone discovered.

Previous history.—The patient, a weaver by trade, and a native of Royton, when five years of age had a severe attack of smallpox, on his recovery from which symptoms of stone made their appearance. His urinary discharges were subject to sudden and frequent intermissions, showed occasionally appearances of blood, and were followed by a stinging pain. These evidences of calculous disease continued with varying intensity, but did not affect his general health until about two years ago, when his disorder assumed a more serious aspect. He now began to lose both flesh and strength. Micturition became very frequent, the urine constantly more or less mixed with blood, and a white, opaque sediment, which he had long observed in the urine, more abundant, and its odour more offensive.

State on admission.—Emaciated almost to a skeleton, he was unable to leave his bed, and suffered from a constant desire to empty the bladder, micturition taking place almost every quarter of an hour, accompanied with intense pain in the loins and lower part of the belly. The patient is small in stature and stolid in temperament.

The chest and abdomen, on examination, were found to be quite free from disease, and all the symptoms to have reference to the urinary apparatus. The urine was charged to a great extent with pus, which, on standing, formed a thick, white, viscid sediment. It was ammoniacal even within the bladder, and a very considerable quantity of albumen was precipitated by boiling and the addition of nitric acid.

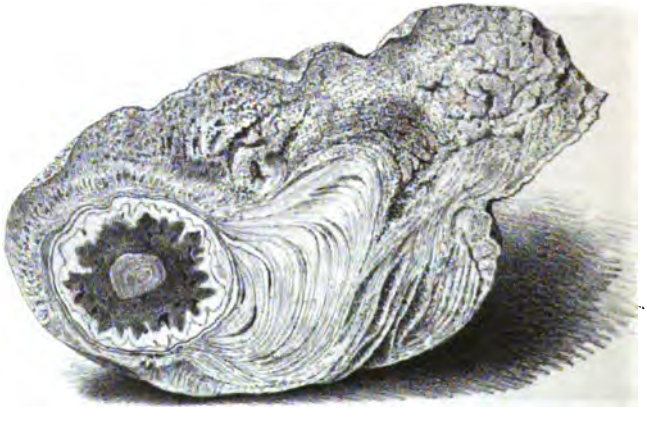
I was invited by Dr. Roberts to see the case, with a view to transferring it to the surgical wards; but operative procedures seemed to be out of the question, until we could relieve the hectic fever and the great debility under which the patient was labouring. It was decided that he should be kept in bed, and have nutritious diet, with eight ounces of wine daily; and that our efforts should be directed to allay the irritation of the mucous coat of the bladder. With

this view the bicarbonate of potash, with opium and large dilution of water, was prescribed. At first the desire to make water was intensified, and micturition rendered more frequent by the increased flow of urine, but in three days the beneficial results of the treatment began to show themselves, and a few days later a considerable improvement was visible. The patient was now able to retain his water for an hour and a half, and, instead of only voiding about a spoonful at a time, could hold half a teacupful in the bladder. The urine, however, still continued albuminous, purulent, and ammoniacal; but, in a month, the patient's strength had so much improved that the question of operation had to be entertained.

Judging from the long period of sixteen years during which the stone had been growing, the grave effects it was producing on the health of the patient, and from the more certain evidence of examination by the sound and the finger introduced into the rectum, I was convinced that the concretion was of large dimensions. The bladder had become so contracted from long inflammation, that only two or three ounces of fluid could be injected into it; the general health was still most precarious; and, to crown the embarrassment, there was still a copious deposit of albumen in the urine. The question that suggested itself was the following: Did this indicate degeneration of the kidneys? If so, any thought of operation must be renounced. To the solution of this difficulty the microscope now afforded valuable assistance, and by this instrument the urine was repeatedly examined to discover whether any fibrinous casts of the uriniferous tubes were present; none, however, were found, nor any reliable indications of renal epithelium. And, on an accurate comparison of the quantity of pus and blood in the urine with the amount of albumen precipitated by nitric acid and heat, we arrived at the conclusion, that the albumen came from no higher source than the bladder itself, and was simply deposited from the liquor puris, and the small quantity of blood always present in the urine.

As the kidneys were considered not to be implicated, an operation was determined upon. For this purpose, the patient was removed to the surgical wards. Wine and nutritious diet were given, and for medicine tincture of hyoscyamus with infusion of buchu. By this means his improved state was maintained; but as no further amelioration could be anticipated as long as the stone remained in the bladder, the calculus was removed by the recto-vesical section on the 17th of December.

Operation.—Chloroform having been administered, the patient was secured as for the lateral operation. As much warm water as possible (about half a teacupful) was injected into the bladder. A scalpel guarded by the finger was then introduced into the rectum, and the sphincter and lower part of the anus completely divided in the direction of the raphe. The staff was now felt for, and the urethra having been opened anteriorly to the prostatic portion, the scalpel was pushed forward along the groove towards the bladder. The finger having been carried into the bladder, and the stone felt, the staff was withdrawn. As was suspected, the calculus proved to be of large dimensions. Accordingly the wound in the prostate was enlarged on its urethral surface with a probe-pointed hernia-knife. By means of the finger the rest of the prostate and neck of the bladder were sufficiently dilated to admit of a pair of forceps. The stone lay sunk in the base of the bladder, stretching its elongated form from the openings of the ureters to the neck of the bladder. On the introduction of the forceps it was found to be impossible to grasp with them so large a stone, as the contracted and indurated state of the bladder prevented the divergence of the blades. A scoop was therefore substituted, but with no better success. The stone could not be dislodged from its original position, having so imbedded itself at the base of the bladder in the mucous membrane, that it felt as if it were encysted. Finding it difficult to lay hold of the calculus in the usual way, I had the screw of a straight-bladed forceps removed, that the blades could be separately introduced, one over and the other below the



stone. The handles being then brought together, and the screw re-inserted, the calculus was grasped and slowly extracted; the operation occupying from ten minutes to a quarter of an hour. Scarcely any hæmorrhage supervened, nor did there appear to be much bruising or stretching of the soft parts. Some fragments were next removed from the rectum, and the bladder well washed out.

The calculus has a rough, uneven surface, irregularly oval, and measures eight inches in circumference in one direction and seven in the other, having a diameter of two and a quarter inches, and being three and a third inches long. It weighs four ounces, six drachms, and twenty-five grains. A section shows that it consists principally of triple and earthy phosphates, in which is imbedded at one extremity a small, round, alternating calculus, about three quarters of an inch in diameter, consisting of lithic acid and oxalate of lime. *See Plate* VII.

At the close of the day of operation the patient was found quiet and comfortable. There were no symptoms of shock; pulse under 100. The bowels had been thoroughly cleaned out before the operation, and opium was now administered to restrain their action and keep the parts quiescent.

December 18th.—Doing well; pulse 90; no signs of undue febrile excitement. Has a long pillow under the knees. All the urine dribbles through the anus. The visible parts of the wound healthy. Ordered, milk diet and four ounces of wine daily.

20th.—Pulse 96; tongue clean and moist; free from pain; appetite good; took a pint of bread and milk for breakfast; felt some desire to go to stool, and was therefore ordered to take food of a highly nutritious character, but in moderate quantities.

22d.—Progressing very satisfactorily; pulse 88. All the urine still passing by the rectum; feels at times as if some would come by the urethra.

27th.—Bowels moved for the first time on the 24th, seven days after the operation, and without the aid of medicine; no incontinence of fæces.

29th.—Bowels moved for the second time this morning; feels an increased desire to pass through the urethra the urine, which still comes away solely per rectum.

31st.—This morning passed about half a cupful of urine by the urethra; bowels moved daily; no incontinence of fæces. All the symptoms favorable.

January 5th, 1859.—Since last report has progressed without a bad symptom; passes water about every two hours by the urethra, and for some time afterwards no urine escapes per rectum. Improves strikingly in appearance, and has lost much of his listless indifference; is gaining flesh.

28th.—The greatest part of the urine now comes by the urethra, and for several days none has passed by the anus, except when the bowels have been moved. An important improvement is perceptible in the character of the urine, which is no longer alkaline and ammoniacal on emission, although on standing it speedily becomes so, as might have been expected from the quantity of pus still remaining. The pus is considerably diminished, and the albumen apparently in the same proportion.

February 18th.—The fistulous communication between the urethra and rectum examined to-day. It is about a quarter of an inch in length, and appears to be situated in the membranous portion. An unsuccessful attempt made to apply the electric cautery, the apparatus not being in working order. The edges were therefore touched with the nitrate of silver.

23d.—The urine contains but little albumen or pus; now gets up daily; strength increasing; wine discontinued, beer substituted. The electric cautery successfully applied to the edges of the fistula, which had slightly diminished in size since last examination.

March 27th.—Still a small quantity of urine passes by the rectum. In other respects appears quite well. The urine now free from pus and albumen. The rectum examined; the fistula barely perceptible; the sound could not be touched through it, but there was felt a small depression

on the rectal mucous surface. To this the electric cautery was applied.

April 5th.—The urine comes almost exclusively by the urethra. Only a few drops pass by the anus during micturition, and of these the patient is quite unconscious, except when feeling with the finger. Is in excellent health, and rapidly gaining weight. Experiences no inconvenience from the operation, and goes about all day assisting the nurses in the wards.

28th.—No signs of the fistula having made their appearance during the last fortnight, he was to-day discharged, *cured.*

Remarks.—From the details of this case, it will be seen that a more unfavorable one for operative interference could scarcely have been conceived. The patient emaciated and debilitated to the very lowest degree; the urine highly purulent, albuminous, and ammoniacal; and the bladder, besides enclosing a calculus of unusual dimensions, in a highly irritable state, and, from disease of its coats, permanently contracted. A careful examination, however, of the urine, led to the belief that none of these symptoms could be traced to the kidneys, for the morphological elements usually accompanying degeneration or disease of these organs were not discoverable. Taking into account the extensive disease of the bladder, of which there was abundant evidence, the apparently large size of the calculus, and the unsatisfactory state of the patient's general health, I was led to consider the rectal operation as the least likely to be attended with fatal results.

Of the several methods of lithotomy, the lateral is undoubtedly the safest where the stone is of moderate dimensions, and as such cases are by far the most numerous, it is seldom necessary to resort to any other operation. The success principally depends on limiting the incision of the prostatic portion of the urethra to its anterior part, which, in the majority of instances, need not be extended, as the greater portion of this structure readily yields to moderate and steadily increased dilatation. Lithotomists have en-

deavoured to determine the precise extent of incision which may be made with comparative safety: thus, Liston recommends that it should not exceed seven or eight lines, others suggest an inch; but, as the prostate varies in size at different ages, I believe it would be a safer rule not to make the incision larger in the first instance than will allow of the introduction of the finger, trusting to dilatation of the remaining portion, which, with slight extension of the incision on the urethral surface of this body, may be readily effected to such an extent as will admit, in the adult, of the extraction of a stone of about an inch and a half in diameter. Beyond this the operation becomes questionable, for a larger calculus cannot, as a general rule, be extracted without extending the wound through the prostate, or occasioning great violence to the neck of the bladder and neighbouring structures. The patient's life thus becomes endangered, either from hæmorrhage consequent on injury to the plexus of vessels which surround the neck of the bladder, and now probably enlarged from disease, or from urinary infiltration into the cellular tissue, or from shock arising from a protracted operation. And, should the patient escape these risks, he may possibly be left with an incurable fistula caused by the sloughing which may arise from the contused and lacerated condition of the soft parts. It has been thought that these dangers might be avoided by breaking up the stone through the wound previously to its extraction; but there is so much difficulty in removing the fragments without injury to the surrounding structures, that it is doubtful if such a proceeding should be resorted to, except in cases where the size of the calculus has not been ascertained before the operation.

It was fortunate that, in the case above mentioned, the indurated and contracted state of the bladder led me to regard it as an unfavorable one for the high operation, for had this mode been attempted, even if the cavity of the bladder had been safely reached, the operation would have been a very tedious and embarrassing one, as the calculus was impacted in the lower part of the pelvis, and possibly

the attempt to extract it might have been a failure, as in a similar case recorded by M. Blandin.

It is unnecessary to dwell on the bilateral operation, a plan now almost entirely exploded, on account of the unfavorable results which have followed its practice, even in the hands of its great advocate, Dupuytren, and other surgeons.[1]

There remained, therefore, only the recto-vesical operation. Several objections have been raised against this method, but the most formidable one is the danger of superinducing an incurable and distressing fistulous communication between the bladder and the rectum. This is not so common a sequel to the operation as is generally supposed, for, according to the statistics of seventy-four cases given by Bourgery,[2] on the authority of Dupuytren, only twelve had fistulas. And in considering this objection, it must be borne in mind that the operation is only *here* recommended where the calculus is of large dimensions, in which case the lateral method (supposing him to escape the dangers of that operation) would expose the patient to equal risk of a fistula from sloughing of the surrounding soft parts.

The other objections—viz., that injury to the vasa deferentia might lead to impotence, that the wound and irritation of the rectum might cause irritative fever, and that recovery is slow—are in some measure counterbalanced by the absence of hæmorrhage, the bladder being reached through the least vascular parts; by the improbability of urinary infiltration, on account of the small amount of cellular tissue involved; by the facility with which a large calculus can be extracted through the wound without tearing or bruising the soft parts; and by the slight degree of shock usually attending this mode of operation. Further,

[1] 'Mémoire sur une manière nouvelle de pratiquer l'operation de la pierre,' Paris, 1836.

[2] 'Traité complet de l'anatomie de l'homme,' vol. vii, p. 281, Paris, 1844.

some of these objections may be obviated in the operation. The proposal of M. Sanson to make the incision into the body of the bladder beyond its neck, has the advantage of preventing injury to the vasa deferentia, but increases the liability to fistula, and that of the worst form, because the communication between the rectum and bladder is direct. This is to a certain extent obviated by making the incision through the neck and prostate, as suggested by Vacca Berlinghieri, the valve-like protrusion of the wall of the intestine cut into below preventing the fæces from passing through the wound, in which case, if a fistula should remain, it will probably only be urinary. But even this may be frequently prevented by not extending the incision through the neck of the bladder, which, I believe, is seldom required, the obstacle to the extraction of large calculi by the lateral method existing more in the surrounding structures than in the prostate, which readily yields to steady and cautious dilatation when in a normal condition.[1] In the case now related, there was abundant proof of this; indeed, a calculus of much larger dimensions than the one described, could have been extracted without difficulty by the same incision.

Amongst the other interesting features in the above case, I would call attention to the slight constitutional disturbance which followed the operation, a circumstance which has been dwelt upon by the advocates of the recto-vesical method, and which it is important to remember, as we sometimes meet with examples in elderly persons suffering from calculus, where, though the stone is not large, so many complications exist, as to render it doubtful whether the lateral operation might not prove fatal, whilst this method could be resorted to with less risk, as a means, if not of a curative, at least of a palliative, character.

[1] Although I have described the operation under the term recto-vesical, it is seen from the description of the case that it would be more appropriately called *recto-urethral*.

ON

DIFFERENT FORMS

OF

PRIMARY SYPHILITIC INOCULATION.

BY

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Received June 28th.—Read June 28th, 1859.

In the year 1856, I published an account[1] of some cases of primary syphilis, in which the test by inoculation gave no indication of their real nature; and I came to the conclusion that the laws which regulate the development of the pustular or suppurating syphilitic sore, do not apply to the primary infecting chancre.

Further experiments and observations appeared to establish the fact, that the morbid action which infects the patient's system is essentially of a different nature to that which has usually been produced as the result of artificial inoculation.

In the year 1857, M. Ricord[2] and his zealous assistants entered upon a formal investigation of the subject of the inoculability of the secretion of an indurated chancre upon

[1] 'British and Foreign Medico-Chirurgical Review.'

[2] 'Leçons sur le Chancre,' p. 267.

the patient who had it. The conclusion which they came to was that an indurated chancre, as a rule, was not capable of being thus inoculated.

Under these circumstances it becomes evident that the product of the indurated sores (those affected with specific *adhesive* inflammation) is not that with which the experiments on syphilisation have been performed; and if it be true that pustular inoculation gives rise to a form of disease similar to itself, and that indurated sores are those alone which may be expected to be followed by secondary symptoms, then it becomes equally manifest that no constitutional syphilitic affection is likely to follow the ordinary practice of syphilisation. Accordingly, if we look through the whole record of these cases, we shall scarcely find one where the practice has been followed by any secondary symptoms attributable to the artificial inoculations. The fear, which so generally prevailed when the practice of syphilisation was first advocated, of introducing fresh syphilitic matter into the patients' constitutions, must now, therefore, be allowed to have been in a great measure ill founded.

Although the product of the specific adhesive inflammation, when fully established, is not, as a rule, capable of being again inoculated with the lancet upon the system which has produced it, yet both experiment and observation concur in proving that it may be so modified by certain actions as to become again inoculable upon the same individual. Under these conditions, however, the inoculations are uncertain in their results, producing very little local irritation, and capable of being transmitted by successive inoculations a comparatively very small number of times.

Case 1.—H. C— came under my care at the Lock Hospital on the 2d of February, 1854. She had a syphilitic eruption upon the skin, and several irritable sores on the inner margins of the labia and perinæum. The inguinal glands were enlarged and indurated.

February 4th. — Several spots were inoculated from angry-looking sores on the margin of the anus and labia.

6th.—Several fresh inoculations were performed from other irritable sores.

11th.—Each point inoculated had produced a kind of pustule. Fresh inoculations were made with the secretion of the original sores, and with that of the artificial inoculations.

13th.—Upon each point last inoculated a vesicle had appeared.

14th.—The secretion of each vesicle had become turbid.

16th.—Fresh inoculations were performed from the sores, natural and artificial, which furnished the largest amount of secretion. Eighteen hours afterwards the inoculated spots were found to be slightly reddened.

18th.—Forty-four hours after the last inoculations, no result was perceptible. Several fresh spots were inoculated with the secretion of the sores near the anus, and with that of the sores artificially produced.

22d.—The last inoculations had produced no effect. The inoculations of the 11th were forming small dry scabs. The eruption has now faded.

24th.—Several spots were inoculated with as much secretion as could be obtained from any of the sores.

27th.—The inoculations last made had produced no effect.

March 2d.—The sores, natural and artificial, had all healed. The patient was discharged as cured, having been under treatment exactly four weeks, and having apparently, during the last two weeks, not been susceptible of any further inoculation by means of the secretion derived from her own sores.

It was at first difficult to say, why in this case some of the inoculations were capable of being reproduced, and some not; why those last made were of a different character from the first; and why the results of all the inoculations disappeared in so short a time.

These questions appear to derive a solution from the fol-

lowing two cases, which show that, although an infecting sore is not capable of being inoculated under ordinary circumstances, yet the same sore will, under a state of irritation, produce an inoculable secretion, and that the effects of the inoculation of that secretion will vary according to the amount of irritation present at the time the secretion was produced.

Case 2.—A lad was admitted into the Lock Hospital on the 29th of July, 1858. He had had gonorrhœa six months previously, but otherwise had been free from any venereal affection until about a fortnight before his admission. He then had a superficial sore behind the corona glandis, which healed in a few days. Two or three days after the first appearance of this sore, a little pimple appeared on the outer skin of the prepuce. This, on the 26th of July, presented all the appearances of a well-marked Hunterian chancre. It discharged from its surface a white turbid secretion. To a portion of this a drop of acetic acid was added, and it was examined by the microscope, and found to contain no pus-globules.

July 27th.—The secretion was inoculated in several points on the patient's thigh.

29th.—This was the day the patient was admitted into the hospital. The secretion from the sore was again examined, and found to contain no pus.

31st.—Several fresh inoculations were made. The sore continued to increase in size.

August 3d.—None of the inoculations had succeeded. The glands at the back of the neck were now enlarged, and the skin presented an incipient syphilitic eruption. A small blister was applied to the surface of the sore.

5th.—A superficial slough had formed on the surface of the sore, which yielded in parts a puriform secretion. This was inoculated upon the thigh in several points.

7th.—The sore now again secreted no pus. Fresh inoculations were performed.

10th.—The sore was dressed twice yesterday with the

sabine ointment, and now yields a copious secretion of pus. This purulent secretion was inoculated in several points on a different part of the thigh.

12th.—The inoculations last made had succeeded. The sore still yielded a copious secretion of pus.

14th.—The inoculations both of the 5th and 10th had now succeeded, but not those of the 7th. They presented the appearance of circular red patches, with some elevation and thickening of the cuticle. In one place there was the appearance of a vesicle, from which a serous fluid exuded. This fluid was again inoculated upon the patient's thigh. The original sore, which had been dressed with water, now again yielded no pus.

17th.—The inoculation from the inoculation had succeeded. It presented the appearance of a red circular patch, with slight thickening of the skin, from which the cuticle was abraded. It had not in the least assumed the appearance of a pustule, nor was anything like pus secreted from its surface. A single pustule, surrounded by very little inflammation, had, however, formed in one of the points first inoculated.

19th. — The inoculations appeared as separate red patches on the skin, which, in these situations, was slightly raised and thickened, but no induration extended into its substance. The solitary pustule which appeared had dried up.

24th.—One of the inoculations first made had a slight tendency to ulcerate; the others were desquamating and losing their colour.

Case 3.—Bridget C—, æt. 17, was admitted into the Lock Hospital on the 26th of August, 1858. She had suffered from a thick yellow discharge between two and three months. This was followed, in the course of as many weeks, by the appearance of two small places on the upper part of the left thigh. These, upon her admission, presented all the characters of well-marked primary indurated chancres in a state of progress.

The surfaces of these sores were covered by a scanty tenacious secretion, in small quantity, which, upon microscopic examination, yielded no pus.

This secretion was carefully inoculated upon the patient's thigh.

August 28th.—The inoculation was repeated. There was, at this time, no indication of the sores having any tendency to heal.

31st.—No result from the inoculations. The two sores had now been dressed for two days with the Unguentum Sabinæ, and yielded an abundant secretion, distinctly purulent. The secretion from each sore was inoculated in several points close together, in two separate places in the thigh.

September 2d.—The inoculations last made have produced the appearance of small incipient pustules in both situations. The secretion from one of these was inoculated in two or three points on the thigh lower down.

4th.—The inoculations from the inoculations had apparently succeeded. One of the inoculations of the 31st of August had produced a small pustule. The others had produced only vesicles. The skin over one of these was broken.

9th.—The inoculations from the inoculations, performed on the 2d of September, have dried up. The inoculations first in order of the 31st of August had entirely lost their puriform character. They now appeared as circular patches, yielding a serous secretion, mixed with epithelial scales. The original chancres were now in process of healing.

In the accompanying Plate VIII, fig. 3 shows the appearances of the inoculations upon the tenth day.

11th.—The inoculations first in order were desquamating, and of a light-red colour. The inoculations from the inoculations appeared as small red pimples, which were gradually losing their colour.

17th.—The original chancres were cicatrized. The inoculations were fading and desquamating.

23d.—The inoculations from the inoculations were still

visible, and appeared as shining scales of discoloured epithelium.

25th.—A few faint secondary spots appeared on the body. The original sores were quite healed, leaving slight induration. The corresponding glands in the groin were still enlarged and hard.

Fig. 4 shows the appearance of the inoculations on the twenty-sixth day.

October 4th.—A fifth drawing was made by Dr. Westmacott, and shows the remains of the original sores, and of the inoculations on the thirty-fifth day, and also the remaining traces of the inoculations from the inoculations.

This patient now left the hospital, but again presented herself on the 8th of October. The inoculations appeared as brown spots, the colour of which gradually faded into that of the surrounding skin.

It is quite possible that the persistence of the inoculations and their peculiar colour in the two last-recited cases may have depended upon the syphilitic diathesis of the patients. But this in no way militates against the fact, that inoculations succeeded at one time, while they failed at another, under precisely the same conditions of the general system.

In Case 1, the inoculations succeeded so long only as the sores furnishing the secretion maintained their irritable character, and failed as soon as this irritability subsided.

In Cases 2, and 3, the sores, the natural secretion of which could not be inoculated with the lancet upon the patient, furnished an inoculable secretion when artificially irritated.

In all the inoculations above recorded, the effects appear to have been in direct relation to the amount of irritation present, and generally in proportion to the puriform condition of the secretion inoculated. It might be supposed, that in the first case the sores ceased to be inoculable because they were in a healing condition, but this would not account for the inoculations produced from

them ceasing to afford an inoculable secretion within four or five days of their first appearance. The inoculation then of the secretion of a sore affected with specific adhesive inflammation may take place, but is not easily performed, when once the patient's system has been affected with syphilis. When successful, the results are very different from those which follow the inoculation of the secretion from naturally suppurating sores. In the latter case each puncture produces a pustule, which, by repeated inoculation, will produce its like a great number of times. In the former, the inoculation as a rule fails, and succeeds only under circumstances of accidental irritation. It then can be repeated a very limited number of times, and the results obtained, even by a number of punctures in one situation (as represented in Plate VIII, figs. 3, 4, and 5), are comparatively of a trifling description.

There is, however, reason to believe that these same inoculations, if practised upon a patient whose system was not already affected with syphilis, would give rise to well-developed primary infecting sores.

The suppurative form of syphilitic inoculation offers one variety which has not hitherto, I believe, been separately described, and which I have ventured to designate as the phlegmonoid variety.

In this affection the inoculated point passes very rapidly into active suppuration. The disease, as a rule, involves the cellular tissue, and the sore produced is surrounded by induration, which, however, generally fades into the consistency of the surrounding parts. The best-marked specimens of this disease which I have witnessed have been followed by an eruption of a brick-red colour, confined to one part of the body, and not unlike in appearance the eruption which occasionally follows the administration of copaiba. After a few days, this eruption subsides of its own accord, does not recur, and requires no specific treatment.

The induration which surrounds this form of suppurative inoculation renders it very liable to be confounded in prac-

tice with the primary infecting sore. The character of the secretion is the only means by which the two can sometimes be distinguished. In the phlegmonoid variety, as in the ordinary suppurating sores, the secretion is distinctly purulent. If a small quantity of the secretion be taken, and a drop of acetic acid added, very little will be seen under the microscope but the distinct nuclei of the pus-globules, and these all very much of the same size and shape.

In the primary infecting sore, on the other hand, the secretion treated in the same way will be found to consist of epithelial débris, mixed, perhaps, with globules of different shapes and sizes.[1]

The phlegmonoid suppurating sore is further characterised by having firm and raised edges. This depends upon the effusion of lymph which circumscribes it. The induration produced by the effusion of lymph is sometimes of considerable extent, but, in general, it wants the peculiar and characteristic hardness produced by the specific adhesive inflammation, and it does not, in general, terminate abruptly, but gradually fades into the consistency of the surrounding parts.

Case 4.—A gentleman presented himself in the end of March last, having a sore on the right side of the frænum. This was surrounded by very considerable induration, which ultimately spread for about three quarters of an inch along the urethra. At one time this induration terminated quite abruptly, and from the feeling alone I should not have been able to distinguish it from the induration which accompanies a primary infecting sore. Upon taking a small quantity of the secretion, which was copious, and adding to it a drop of acetic acid, very little could be seen under the microscope but the nuclei of the pus-cells, well formed, and all of the same size.

This disease appeared two days after exposure, and was

[1] In examining the secretion of syphilitic sores, it is essential that they should not have been subject to any previous artificial irritation.

followed in three weeks by an eruption confined to the left arm, and presenting no specific character. The sore became deeper and deeper, and ultimately opened on the opposite side of the frænum, discharging a considerable quantity of pus from both openings. This patient, who had picked up some medical information in Paris, would scarcely be persuaded, finding the extent of the induration and seeing the eruption, that his system was not affected. He has, however, recovered, and remains well, without any sign of constitutional disease, and without any specific treatment that could have prevented the appearance of secondary symptoms.

Case 5.—A gentleman, who had for four years been under the care of some of the first medical men both here and upon the Continent, and who had fairly exhausted all the ordinary modes of treatment, became desirous, as a last resort, to try a modified course of syphilisation. He had at the time a deep copper-coloured eruption, considerably raised in parts, upon the head, face, and neck; with patches of a similar kind on various parts of the body, and a large ulcer on the foot.

On the 13th of April, some pus was taken from the last patient (Case 4), and inoculated in three points upon this patient's arm.

April 16th.—Three inflamed pustules had formed. Two fresh inoculations from the secretion of one of these pustules were made upon the upper arm.

19th.—The inoculations of the 13th had all become surrounded by induration, which became less and less from the centre towards the circumference. They were all broadly fringed with a halo of a pinkish-red colour, which gradually faded into the colour of the surrounding skin. The diameter of this redness was about the size of a sixpenny piece. The inoculations of the 16th had become pustules, not surrounded by much redness. Two inoculations with the secretion of the inoculations of the 16th were performed on the left side of the abdomen.

22d.—The hardness around the inoculations first made was less. These presented the appearance of circular ulcers, with raised edges, affording an abundant secretion of pus. The inoculations second in order were surrounded by induration, but not to the same extent as the first. The inoculations of the 19th had become well-formed pustules, surrounded by redness to a very slight extent. Two fresh inoculations were made on the abdomen, below those last mentioned.

26th.—The inoculations of the 16th were now surrounded by more general diffuse induration than those first made. Two fresh inoculations were made below those of the 22d.

28th.—The inoculations of the 26th present the appearance of very small pustules (forty-eight hours). The inoculations of the 19th discharged a large quantity of pus, and were surrounded by considerable irritation. The inoculations of the 22d were much less inflamed; those of the 26th still less. Two fresh inoculations.

May 2d.—The original inoculations on the arm were still very irritable. They appeared as circular ulcers, with slightly undermined, but raised edges, surrounded by a red areola. The inoculations made on the abdomen were each in succession less in size, with less inflammation and less secretion than those of a previous date. Two fresh inoculations with the matter taken from the previous inoculations which furnished the largest amount of secretion.

6th.—The inoculations last made appeared as very small irritable points, affording scarcely any secretion. The inoculations on the arm were still red, inflamed, yielding an abundant secretion of pus, and presented something the appearance of boils after they have been some days opened. The inoculations first made on the abdomen had the same character. The edges were raised, red, and terminated quite abruptly. The secretion of pus from these was most abundant. Two fresh inoculations from the sores on the abdomen yielding the largest amount of secretion.

13th.—The syphilitic eruption on the face, neck, and

head now showed a decided improvement. The ulcers resulting from the inoculations first made were nearly three fourths of an inch in diameter.

17th.—An eruption of a brick-red colour, somewhat resembling nettle-rash, or the eruption which follows the use of copaiba, appeared on the upper part of the left thigh. It was raised above the surface, with small intervals of nearly healthy skin. It was accurately defined, and strictly limited to the upper part of the thigh, and was totally unlike any eruption which the patient had previously had.

21st.—The eruption had extended, still with a defined outline and minute intervals of comparatively healthy skin. Portions of it were of a more livid hue than before.

23d.—The accompanying drawings, made this day by Dr. Westmacott, represent the appearance of the inoculations (with the exception of those second in order), and also the eruption which resulted from them, and which, in my opinion, was not of a syphilitic nature.

Plate IX, fig. 1, represents the inoculations first made as they appeared on the forty-first day, still in a state of progress.

Fig. 2 represents the third set of inoculations, as they appeared on the thirty-fifth day of their development; the fourth set on the thirty-second day; the fifth set on the twenty-eighth day; the sixth set on the twenty-fourth day; the seventh set on the twenty-second day; and the eighth set on the eighteenth day.

At this period the eruption had lost much of its peculiar brick-red colour, and had assumed generally a livid hue.

27th.—The eruption of the thigh had much faded. The surface over which it extended was now of a more livid hue, but there were still visible a few scattered points of a brick-red colour. The margin of the affected skin was perfectly well defined, and the skin in the neighbourhood had undergone no alteration in any respect. The inoculations on the arm showed a slight tendency to contract. Those subsequently made also show a disposition to heal. The syphilitic eruption on the face and other parts had improved in character.

The patient attributed the improvement to the inoculation, and remarked that some patches of eruption on the arm and body, that no previous treatment "could move, have now altered their character."

June 4th.—The eruption on the thigh was dying away.

11th.—The first inoculations were now covered with thickened skin. They were still surrounded by induration, which gradually diminished to the consistency of the surrounding parts. The inoculations second in order had fairly healed. The inoculations last made, which a few days before had nearly healed, now assumed a slight tendency to spread. There was a marked improvement in the old syphilitic symptoms. Patches of eruption had in a great measure lost their colour, and healthy portions of skin appeared upon their site. The ulcer on the foot, which had remained unhealed for between two and three years, had much improved. The eruption on the thigh (consequent, as I believe, upon the inoculation) had entirely disappeared without treatment, but the skin over which it had spread was of a slightly purple colour.

This patient was now recommended to go into the country, and shortly after left England for the Continent. The face was then quite clear, but some patches of eruption still remained on the body. From the commencement of the inoculations no internal treatment was adopted, which could have influenced the course of the disease.

14th.—The patient (Case 4) from whom the pus was taken for the purpose of inoculation presented himself. His sore had healed a fortnight ago, leaving induration under the skin, but not in the skin. He was quite free from any appearance of constitutional disease.

The character of the inoculated sores in the above case accurately resembled that of the sore from which the secretion was originally taken, allowance being made for the difference of situation. In both cases the sores were surrounded by induration; in both they yielded a copious

secretion of pus; and in both they were followed by an eruption confined to one part of the body, and disappearing permanently without any specific treatment.

The nature of the secretion in the diagnosis of primary venereal sores is of considerable importance. It is, perhaps, the only means by which a suppurating sore of the phlegmonoid variety can, in some instances, be distinguished from an infecting chancre. In both cases the induration is caused by the deposit of lymph; and although this deposit is generally much more accurately defined, and terminates much more abruptly in the infecting chancre, yet such is not always the case. The morbid actions are, however, essentially different. In the one case the lymph is converted into pus; in the other case it is thrown off in the débris of a scanty discharge, or remains until it is again taken up into the system. The first is a form of suppurative, the latter of adhesive inflammation. It is true that any sore, if exposed to artificial irritation, may readily yield a puriform secretion; but in any doubtful venereal case, if the sore be dressed with water dressing for a day or two, the nature of the discharge will then become apparent.

During parts of the years 1855-6, tables were kept at the Lock Hospital, and for some time at King's College Hospital, of the nature of the discharge presented by venereal sores upon microscopic examination. Among these cases were ninety-five presenting all the ordinary characters of primary infecting sores. In none of the instances was the secretion (care being taken to avoid causes of accidental irritation) purulent. The secretion consisted generally of epithelial débris floating in a serous fluid, or of globules of various shapes and sizes, which did not, upon the addition of acetic acid, yield the well-defined nuclei characteristic of pus.

In a considerable number of cases the secretion appeared to consist of nothing but epithelial débris and serous fluid, and in some there was no fluid secretion at all.

During the same period, the tables referred to show

seventy-three cases of secondary disease resulting from primary indurated sores. On the other hand, during nearly the same time, one hundred cases of syphilitic sores were noted in which the secretion was, upon microscopical examination, judged to be distinctly puriform.[1] These cases were, as a rule, treated without mercury; and in two instances only did the patients, as far as I know, return with secondary symptoms. In both these cases there had been more than one source of infection, so that the suppurating sore may have made its appearance either before or after the infection of the general system.

If it be true that suppurating syphilitic sores are readily inoculable with the point of the lancet upon the patients who bear them, and that the indurated sores (those affected with the specific adhesive inflammation) are, as a rule, not capable of being thus inoculated, it would then appear that the suppurating and indurated sores have often been mistaken for each other. In Dr. Sperino's experiments on syphilisation, for instance, we find it frequently asserted that the secretion from an indurated chancre was inoculated, and produced the specific pustule. These assertions are so much at variance with more recent experience, that the only conclusion that can be drawn is that these so-called indurated sores belonged in reality to the phlegmonoid, or some other variety of suppurating syphilitic sores.

[1] Many doubtful cases were excluded.

DESCRIPTION OF PLATE VIII.

Fig. 1.—Indurated sores in process of healing.

„ 2.—Original sores, covered with epithelium.

„ 3.—Inoculations from sores artificially irritated, showing the appearance on the tenth day.

„ 4.—The appearance of the inoculations on the twenty-sixth day.

„ 5, A.—Original sores healed.
B.—Remains of inoculations on the thirty-fifth day.
C.—Remains of inoculations from inoculations.

Fig. 1.

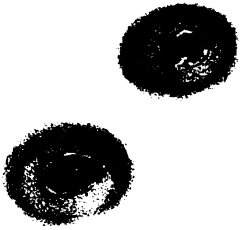

Fig. 3.

Fig. 4.

Fig. 2.

DESCRIPTION OF PLATE IX.

FIG. 1.

A.—Inoculations of secretion from a suppurating sore, as they appeared on the forty-first day. Phlegmonoid variety.

FIG. 2.

B.—Third set of inoculations, represented on the thirty-fifth day.

C.—Fourth set, on thirty-second day.

D.—Fifth set, on twenty-eighth day.

E.—Sixth set, on twenty-fourth day.

F.—Seventh set, on twenty-second day.

G.—Eighth set, on eighteenth day.

H H.—The eruption on the thigh, as it appeared on the eighth day, being the forty-first from the first inoculations.

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